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THE CIRCULATORY REGIME OF GLAUCOMA.

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This is a discussion of the relation of the circulation, especially the venous circulation, to the etiology of glaucoma. Blood pressure, general taken in the brachial artery, and local in the arteries of the retina and choroid, systolic and diastolic and the changes of venous blood pressure are analyzed in relation to the intraocular tension. It is suggested that the most general cause of glaucoma is the narrowing of the venous blood channels caused by inflammation.

So much has been written about glaucoma that we feel some hesitation in increasing an already enormous bibliography. Yet, if we are to establish the pathogeny of this affection, which still seems to be obscure, it is only by accumulating facts that a solution may be hoped for. In studying the state of the intraocular circulation in glaucoma, we do not pretend to impose a vascular theory of the ocular hypertension. The facts we are about to report, the acquisition of which has been rendered possible by the method of measuring the local blood pressure, are, for the most part, already known. But these modifications, functional or anatomic, of the ocular vessels have been diversely interpreted. They have been most often considered as being the cause rather than the effect of the ocular hypertension. We shall endeavor to show that the opposite proposition can be upheld, and we shall thus study, without excluding other mechanism, the rôle of the ocular circulation in glaucoma.

The habit of using the term glaucoma to designate a pathologic hypertension has prevailed. We may deplore it, but we must submit to it on condition that we explain ourselves. We shall therefore specify first of all, altho we consider that there is a relationship between all pathologic hypertensions, that we shall take as a typical instance, chronic glaucoma, with variable, but permanent hypertension.

The frequency of vascular disturbance in glaucomatous patients is well known; all other writers on the subject have spoken more or less of sclerosis, or cardiac or renal lesions. C. Charlin is said to have met with them in 90% of cases, and Kümmel, in practically the same proportion. But it is evident that all cardiac and renal patients are not glaucomatous; if vascular lesions can intervene in glaucoma they must therefore have affected the intraocular system of blood vessels.

As for arterial hypertension, it is indisputable that it is frequent. But let us beware of being dogmatic. In the same way as not all cardiac or renal patients are suffering from hypertension, there exist glaucomatous patients possessing a low or normal general arterial pressure (Terson and Campos, Kraemer). However, most of the cases of chronic glaucoma with constant ocular hypertension are accompanied by a high arterial pressure revealed by the armlet.

In a normal eye the tonus is incontrovertibly influenced by the pressure of the blood which circulates in the vascular system of the organism. Hence, no doubt, proceed the daily modifications of the ophthalmotonus. But this influence does not exceed certain limits, and we meet with patients in whom the sphygmomanometer indicates a systolic brachial pressure of 300 mm. Hg. without any suspicious ocular sign.

We know today that the principal cause of the arterial hypertension is the sclerosis of peripheric arteries, to which is added a more or less pronounced spasmodic state. (It is this spasm which would yield to the in-

fluence of high frequency currents). This degeneration is found also in the ocular arteries, their walls lose their elasticity, the lumen shrinks, and parallel with the volumetric diminution of the ocular blood column appears a lowering of the ocular tonus[1]. Let us not be astonished therefore if certain cardiorenal patients have a low ophthalmotonus, in spite of an alarming brachial pressure. We admit with Wessely[2] that in aged persons, a low ocular tension means a bad peripheric circulation. The state of sclerosis of the ocular arteries, known since the time of v. Graefe, does not appear, therefore, susceptible of causing an elevation of the ocular tension, but on the contrary of lowering it. If the circulation has any influence in the pathogeny of glaucoma, let us say at once, it is a disturbance in the outward flow of the blood, much more than a disturbance in the arrival of the blood, that must be incriminated.

THE INTRAOCULAR CIRCULATION IN NORMAL EYES.

However near they may be to each other, the two intraocular systems of vessels, choroidal and retinal, have very different functions. One, specially charged with assuring the visual function, is a very autonomous system, with one entrance and a single exit, no anastomoses, no important dilation possible, either of the capillaries or of the veins. The other, the choroidal system, which not only assures the nutrition of the outer layers of the retina, but is also charged, without any possible doubt, with a role, the importance of which has never been denied, in the regulation of the ocular tension, is a system widely open with its four great posterior channels, and with its smaller anterior channels, constituting with its trunks and capillaries, by itself, almost the whole thickness of the choroid, it is eminently dilatable. We shall endeavor to picture to ourselves what may be in the normal state, and in glaucoma, the regime of these two circulations. One, the retinal circulation, can be examined directly and is perfectly known to us. As to what goes on in the choroidal circulation, we can get some idea, by its probable analogy with the retinal circulation and by the study of the oscillations of the needle of the tonometer.

We know that in a person, whose ocular tension and general arterial pressure are normal, the pressure in the branches of the central artery on the papilla is from 30 to 35 mm. Hg. minima, and about 70 maxima. Measured at the same point, that is as near as possible to its outlet from the eyeball, *the venous pressure is in equilibrium with the ocular tension.* It slightly exceeds the ophthalmotonus during the diastole and exceeds it very slightly during the systole. Let us not be astonished at this fact! The venous pressure may in a normal circulatory condition be somewhat less than the tension of the medium by which the vein is bathed; the flattening of the vein does not follow, as might be at first believed. Two elements intervene to keep it open: on the one hand the pressure itself, which is exerted in the interior of the vein, and on the other the resistance of its wall. It is thus seen that the ocular tension may, without disturbing the local circulation, be superior to the retinal venous pressure, on condition that it only exceeds it by very little.

The choroidal system of blood vessels cannot, as a rule, be examined directly. We may guess, however, that the proximity of these vessels, their common origin, their practically analogous diameter, at least as concerns the arteries, insures their having a similar circulatory regime. Moreover, it is not exceptional, thru an atrophied spot in the choroid, to see distinctly small choroidal arteries. They react generally by pulsation on compression of the eyeball, and we have been able to note in this manner *an absolute similarity between the retinal and choroidal arterial pressures.* The same result is reached by Seidel by a somewhat different method than the one we employ. Thru a small glass vessel, in the interior of which he causes the pressure to vary, observing the perforating branches of the anterior ciliary arteries, he notes the appearance and disappearance of the ar-

terial pulsation[3]. These measurements lead him to admit that the pressure in the ciliary perforating branches is from 35 to 45 minima and from 55 to 75 mm. Hg. maxima. If we take into account the inevitable divergences existing on the same subject (likewise for the same artery) between results obtained by two different methods, we must admit that Seidel's figures confirm our data that the arterial retinal and choroidal pressures are practically the same. Oscillometry of the eye by means of the tonometer leads also to the same result.

It is very probable that the same analogy exists for the venous pressure between the two systems. No doubt, owing to the four great vortex veins mentioned earlier, the choroidal system is less shut in than the retinal system; and this might cause a lower pressure were not the arterial inflow considerably greater. Seidel, measuring, by the method he applied to the arteries, the pressure in the ciliary veins considers that it is from 10 to 14 mm. Hg. Let us not forget moreover that between a vein held in a closed cavity and a vein running freely under the conjunctiva, there is necessarily a difference in pressure; so long as the walls are supported and do not let themselves be forced, the interior pressure remains almost the same as the capillary pressure. But when a vessel becomes free again its walls may dilate and at the same time the pressure, in a vessel which has become larger, may fall. Seidel's figures are therefore very plausible and lead us to admit that, like the retinal pressure, the pressure in the choroidal veins before they leave the eyeball is practically in equilibrium with the ocular tension.

These few points of normal vascular physiology having been recalled, what do we find in the intraocular circulation in glaucoma? The retinal system will give us much more information than the choroidal system.

OCULAR BLOOD PRESSURE IN GLAUCOMA.

In considering the arterial pressure of the retinal system in glaucomatous patients, we must first of all not forget the predominant role of the general pressure.

The figure even of the arterial retinal pressure is relatively unimportant; it is more important to know whether, in hypertensive states, the normal ratio is preserved between the general and local arterial pressure. When a single eye is in a state of hypertension, or when both eyes are so but unequally, it is well to compare the retinal pressure of both eyes. The result of these comparisons is to show us, that every time there is ocular hypertension we meet at the same time with a relative local arterial hypertension. It is not less interesting to note that if either by medical treatment or by operation, the ocular tension has been restored to normal, the arterial pressure is also seen to fall. Let us bear in mind this fact: the rise of the minimum retinal arterial pressure, sometimes slight, sometimes great, is the rule in the majority of cases of ocular hypertension.

In glaucoma, what is the pressure in the retinal veins?

Sometimes it exceeds the ocular tension; in the great majority of cases, it remains, as in the normal state, in equilibrium with it. The proof of this rise of pressure in the interior of the vein is that its walls do not collapse. We have already seen that this falling-in could be avoided only if the venous thrust, made up of the venous pressure and the resistance of the wall, (a very slight resistance indeed) was able to counterbalance the pressure of the surrounding medium.

The choroidal veins are, in glaucoma, entirely hidden from our examination. But two systems, issuing from the choroidal system deserve to hold our attention, the *anterior, subconjunctival-ciliary system, and the system of the iris.*

As we are speaking to oculists, it is unnecessary to describe the abnormal state of these vascular subconjunctival trunks, which so often appear dilated in glaucoma. In the most typical cases we may speak of a resemblance to the head of Medusa. The nature of these vessels has been much discussed; altho from their appearance, their sudden emerging on a level with the scleral hole, they have often been described as perforating ciliary arteries (since Leber does not admit

the existence of anterior perforating veins). It can be affirmed that the greater part of the dilated vessels are veins. This is true, above all, for the vessels which have their point of perforation *close to the limbus.*

The best way to recognize their nature is to press on them with the finger, crushing them thru the lid from the center to the periphery; by watching the way in which they fill themselves, the movement of the blood is recognized; in most of the small vessels in question, the repletion takes place from the cornea, towards the periphery. But the blood column which, in a normal eye forms slowly, is reconstituted in cases of ocular hypertension with such rapidity, that it is sometimes difficult to perceive the direction of the movement. This is indisputably the result of an *increased venous pressure.* Moreover, we see much finer vessels which fill themselves from the periphery to the center, thus showing their arterial nature. But why do these veins appear to us thus dilated? We have already had occasion to say why. Compressed in the interior of the eyeball, the venous pressure being equal to the ocular tension, on leaving this enclosed medium they are dilated by their internal pressure and by the excentric thrust, which acting on walls no longer supported, enlarge and dilate their diameter. The dilatation of the veins of the conjunctival system denotes a hypertension of the veins of the uveal tract.

Not less interesting is the *system of the iris,* which becomes in certain cases of ocular hypertension, of considerable importance. To tell the truth, it is rare to find a marked dilatation of the subconjunctival system without the system of the iris being also affected. Very often (and here lies one of the dangers of iridectomy in cases of great hypertension), we see on the anterior face of the iris certain isolated vessels appear, which indicate already an intense congestion. In certain cases, particularly in forms of long standing and yet more in those which follow thrombosis of the central vein, we remark on the anterior face of the iris a remarkably developed system of vessels. These are *veins,* for it is a certain fact that the compression of the eyeball does not bring about the pulsatile reaction so characteristic of arteries, it effaces more or less completely the vessels and that is all. In accordance with these ideas one of us has published the following very demonstrative case[4].

"A man suffering from secondary glaucoma, after a thrombosis of the central vein, showed a very important venous network on the anterior face of the iris. One of the main vessels of the venous network burst, partly flooding the anterior chamber, the pressure lowered immediately and fell from 50 to 35 mm. Hg."

The appearance of the venous network of the iris in cases of high ocular hypertension is another absolute proof of a serious disturbance in the return circulation of the anterior ciliary domain; only a considerable venous hypertension can explain the abnormal aspect of these vessels.

To sum up: *The vascular pressure in states of ocular hypertension can be characterized in the following manner: moderate rise in the arterial pressure, considerable increase in the venous pressure, both in the choroidal and retinal systems.*

The question now presents itself: Should this increase in the venous pressure which we note in the intraocular vessels be considered simply as a consequence of the ocular hypertension?

It is evident that as the pressure in the interior media rises, circulation remains possible only if automatically the venous pressure can follow the rise and remain in equilibrium. As far as concerns the retina, we know that neither the obliteration of the central artery, which lowers to 0 the venous pressure, nor thrombosis of the central vein, (at any rate in the beginning) bring about any modification of the ocular tension. Does the same thing happen in the choroidal circulation? That is the knotty point of the question.

The mere consideration of the anatomy of the systems, makes us foresee already that their obstruction must have different results. If we add up the volumetric total of the retinal veins, we find an almost insignificant figure as compared to that of the chor-

oidal veins; moreover, these retinal veins surrounded by the optic fibers can dilate but very slightly if we compare them with the possible distension of the choroidal system. For the choroid is in fact merely an extremely ramified venous mesh, and it is impossible to conceive its volumetric expansion without there resulting a compression of the ocular media.

A priori, it seems therefore logical to admit, that if in the retinal and choroidal systems, the venous pressure is high, the rise of the venous pressure has in the retina neither the value nor the significance of the same rise in the choroidal system. *The latter alone is capable of playing an active part.* As for the rise of the arterial pressure it may, as we shall see later, be due to the slackening of the venous flow.

What strikes the investigator most, when he makes up the balance sheet of his knowledge, is the importance of what remains to be known. Side by side with definite zones are revealed zones of shade, in which nothing clear can be held to be proved. Is this the case? The part we attribute to the choroidal circulation in the modifications of the ocular tonus is not a mere opinion. Without pretending that it plays an exclusive part, we believe we may affirm its importance from a number of presumptions. Let us set forth some of them:

(a) The obstruction of the vortex veins provokes a considerable hypertony. The obstruction of the anterior venous vessels (anterior ciliary veins) has a like effect.

(b) In an animal killed by bleeding, perfusion (artificial circulation) restores the ocular tension to its primitive level[5].

(c) Compression of the carotid causes hypertony.

(d) Retrobulbar injection of adrenalin acts in a similar way, (Fromaget[6]); subconjunctival injections likewise, but in a lesser degree.

(e) Per contra, injections of irritating substances (NaCl, sublimate, etc.) provoke hypertony by vasodilatation[7]. (Wessely).

(f) Inhalations of amyl nitrite, a substance which dilates the capillaries, causes an increase in the ophthalmotonus (Wessely, Leplat, Bailliart and Bollack, etc.)

(g) Contusion causes (by choroidal hyperemia) a hypertension, not only of the contused eye but also of the other eye[8] (Leplat).

(h) Section of the vasomotor (sympathetic) nerve causes a rise in the tension, its excitation a lowering of the ophthalmotonus concomitant with the state of the vessels[9] (Magitot and Bailliart).

By adding to these facts the circulatory modifications in the iris and in the episcleral veins of glaucomatous patients, we obtain a sum of probabilities which no independent mind can reject.

VENOUS LESIONS IN GLAUCOMA.

In glaucoma, the modifications of the local circulation are of such frequency that we cannot but be struck by their coincidence. Many will reply that these circulatory phenomena are well known, that they are engendered by the increase of the ocular tension, which is itself caused by the hypersecretion of the aqueous humor, or by its retention. According to their reasoning, which we already recall, the increase of the ophthalmotonus causes a difficulty in the return circulation, and some obstacle to the venous circulation tending to provoke blood stasis, therefore, there is no reason to be surprised either at the dilatation of the central vein of the retina or the tortuosity of the episcleral veins.

This explanation seems irrefutable. Its appearance of logic satisfies the immense majority, who firmly believe that the hypertony of glaucoma is due to a sclerosis of the angle, a belief chiefly based on the indisputable therapeutic results of sclerocorneal trephining and of the filtering cicatrices.

It seems audacious to contradict so universal a belief, and no less rash to think that those whose minds are made up are likely to change their opinion. But for the ophthalmologist who is still willing to consider these things impartially, and also for those who, having no opinion, are seeking on which side lies truth, we wish to point

out that the reverse reasoning cannot be set aside.

It is admitted that ocular hypertony engenders the venous stasis; we think that the proposition can be reversed, and that we can say that it it is the venous stasis which engenders the hypertony. This is not a sophism. The clinical facts and the anatomic observations give as much likelihood to, and show even more probabilities in favor of this latter proposition.

A similar reasoning has been upheld on several occasions chiefly before the extraordinary vogue of Leber's ideas; Birnbacher and Czermak[10], Stirling[11], Roser[12], von Schoen[13], Koster[14], Heerfordt[15], etc., etc. Elliot, in his excellent book, does say that the theory of glaucoma as a vascular disease comes back sporadically, but he adds that the authors are unable to bring forward any fresh proofs, and that they take the effects for the cause. Is this a final judgment? No, since the discussion opened 50 years ago, it is not yet closed, but only a protagonistic opinion on an operation the beneficent action of which is explained by "the opening of the angle of discharge."

To form an opinion, it is necessary to open the anatomic history of glaucoma and to study the facts without allowing ourselves to be disheartened by the dryness of the protocols or to be influenced by the suggestions of those who have transcribed them. All too often the authors make the mistake of leaving in the background whatever is at variance with their convictions, a method which has alas only too often envenomed the debate.

This history is, however, rather small. We must indeed set aside all that concerns eyeballs suffering from a disease of long standing. Every one knows that the opportunity of examining recent glaucoma depends on the chance of a decease caused by an intercurrent disease.

Birnbacher and Czermak[16] made recorded anatomic examination of 7 eyeballs, of which the last four can be set aside as being cases of long standing.

Case 1. A glaucoma evolving since 6 months, enucleation after inflammatory attack, the sight then being much reduced; dilated pupil, very high tension. Examination shows an inflammation of the whole uveal tract with a maximum in the anterior segment. The veins, particularly the medium and large ones, are gorged with blood and surrounded with leucocytic sheaths. The inflammatory foci occur at the trunk of the vortex veins and their branches, others are to be seen also around the ciliary nerves. The perforating vessels are dilated and surrounded with inflammatory cells. The iris is atrophic. A few adhesions exist, between the face of the iris and the pectinate ligament, with leucocytic infiltration around Schlemm's canal and masses of pigment. Retina somewhat degenerated, pupil crater like.

Case 2. An amaurotic glaucoma evolving since two years, not very painful, cloudy cornea, motionless pupil, great hypertension, no cataract, episcleral veins dilated. The anatomic examination shows a diffuse chronic inflammation of a part of the choroid, a very evident endophlebitis in the vortex veins, of which two were obliterated. Iris partly atrophied with thickening of its ciliary portion. In the vitreous and in the anterior chamber, cells containing pigment are found. The episcleral vessels are dilated and surrounded with leucocytic streaks.

Case 3. No clinical information. The examination is characterized by a diffused inflammation of the whole uveal tract propagated to the episclera and to the anterior ciliary vessels and to Schlemm's plexus. The vortex veins are the seat of endo- and periphlebitis.

(*b*) Birnbacher[17] reports this case.

Woman, aged 54, in hospital for a fracture of the ribs. One eye suffers from complete glaucoma. The other eye previously healthy is suddenly attacked. Ciliary pains, mydriasis, hypertony, etc. A few days later, the symptoms abate, sight improves. Death from pneumonia intervenes a week after.

Choroid thickened. Arteries empty, veins and capillaries gorged with blood. Perivascular infiltration of mono- and polynuclear leucocytes: here and there inflammatory foci in the adventitia of

the vessels. Schlemm's canal free. Iris infiltrated and thickened in its upper half. Angle of the iris obliterated only in one sector. Corpus ciliare slightly hypertrophied. Crystalline protruded.

Vortex veins specially studied. In three branches of the supero-internal vein was to be seen an accumulation of leucocytes, for more than 1/3 of their length. Other branches of the supero-external vein showed endo- and periphlebitis, with thickening of the walls. Retina with cystoid degeneration in its anterior segment.

(*c*) Levinsohn[18] reports the case of an aged woman who died a few weeks after entering the hospital as a lunatic. She was suffering from a chronic inflammatory glaucoma, notably relieved by the use of eserin. At the time of her death there was little hypertension. The anatomic examination shows highly developed episcleral vessels, gorged with blood mixed with numerous leucocytes and pigmentary fragments. Iris slightly sclerosed, sphincter atrophied, likewise the stroma.

Among the vessels some are greatly dilated, others sclerosed with walls much shrunken; in the angle are seen inflammatory streaks and cells charged with pigment. Corpus ciliare abnormally voluminous and yet sclerosed. Its vessels as well as those of the ciliary processes showed a massive thickening of their walls. All were full of blood and leucocytes.

The choroid was greatly hypertrophied, without any particular modification. The vortex veins seemed to be pervious, but in certain places showed endophlebitis, which made them shrink in a manner which cannot but be taken into account. The spaces around them were like all the choroidal vessels, surrounded with pigment. There existed also lesions of the vessels of the optic nerve. The central retinal vein was almost completely obliterated behind the disc. But the retina was normal, save for a peripheric vascularisation and a few fine hemorrhages.

(*d*) Hussel[19] reports anatomic examination of an eye in which glaucoma had been following its course for 4 years, and had ended by an acute inflammatory attack. The lesions were characterized chiefly by atrophy of the iris, of which the vessels are in a state of hyalin degeneration and have almost no more lumen. The corpus ciliare is hyperemic with signs of chronic inflammation and edema, the ciliary processes are hypertrophied, the scleral perforating vessels are surrounded with leucocytes, the episclera is infiltrated with the same cells, especially around the vessels; small hemorrhages are noted. Schlemm's canal is full of blood, but it is shrunken in a few places. Its walls are infiltrated with round cells and with pigment.

(*e*) Thomsen[20] records the case of a man aged 70, suffering from complete glaucoma of the left eye and chronic glaucoma of the right eye. A few weeks before his death the right eye had been painful and injected, but pilocarpin had produced improvement. Four days before death from pneumonia the eyes were "white." Fixation of eyeballs one hour after death. The anterior chamber is slightly diminished, the angle widely open, except on the nasal side where pigmentary masses are noted. Pectinate ligament normal, with grains of pigment which are also found in the canal of Schlemm and even in the ciliary muscle. Iris slightly atrophic, scleral vessels without cellular infiltration; broad posterior synechia on the nasal side. The sclerotic appears to be normal. The choroid shows arteries whose walls are very thick. The veins are gorged with blood. The vortex veins, which were all found again in the serial sections are the seat of modifications. The upper nasal vein shows a process of obliterating endophlebitis, with cellular and periphlebitic proliferation and pigmentary sheath. Another smaller vein running upwards shows likewise symptoms of perivasculitis with streaks of pigment. In the retina, the visual cells are well preserved, no hemorrhage, normal layers except for a peripheric cystic degeneration. Papilla without excavation.

(f) Hanssen[21] reported the case of a woman aged 68, suffering from chronic glaucoma. Had suffered a short while before her death from cardioaortic accidents and an attack of glaucoma re-

lieved by eserin. Eyeballs fixed and cut into sections.

The anatomic examination shows perivasculitis of the episcleral vessels. Leucocytic infiltration in the corneal layers in the neighborhood of the limbus. Grains of pigment in the deep corneal layers.

An anterior synechia. Grains of pigment around the canal of Schlemm and the perforating veins. The perforating veins are often replaced by foci of leucocytic infiltration. In the iris, leucocytic infiltration of the superficial layer. Capillaries dilated. Grains of pigment free or intracellular. Profound modifications of the pigment epithelium, the cells of which have lost their limits and the nucleus of which is modified in its form and in its tinctorial affinities.

The retina contains small hemorrhages. Vascular sclerosis. A slight aneurism.

The choroid shows a disseminated infiltration of round cells and a few leucocytic islets, the vessels have a sclerosed wall and their lumen is shrunken by an endothelial proliferation.

(*g*) Rönne[22] cites a case of simple chronic glaucoma, which had evolved without pain or vision of colored circles. The tension of this eye (the left) was 40 mm. Hg. but the application of pilocarpin continued for a month, during his stay in an urologic ward, had reduced the tension to 22 mm. which figure was noted two days before death from an attack of uremia. The anatomic examination of this eyeball, which had been preserved under favorable conditions, shows a normal cornea and anterior chamber. In the iris there are no signs of anything unusual, unless it be small cellular masses in the superficial portion of the stroma. The angle is completely free. The canal of Schlemm empty. In the pectinate ligament there are signs of sclerosis with grains of pigment and pigmented cells.

The sclera is normal. In the choroid nothing abnormal. In the sections unfortunately only one vortex vein could be found, the wall of which is normal. Nevertheless at the level of the scleral crossing, this vessel shows a concentric stricture at its lumen which diminishes it by about 1/3. In the retina the vessels are normal. In the optic nerve the artery has no lesion while the vein is extremely shrunken by fibrous tissue. Other anatomic examinations might be found in the literature of the subject, such as the three examinations of Bartels; but we have selected the cases as they stand.

These anatomic summaries call for an explanation from the point of view of the quality of the organs examined. All, except one, (the case of Rönne) had been the seat of inflammatory phenomena, either old or recent. Should we for that dispute their demonstrative value? We know that the majority of cases of primary chronic glaucoma have attacks ("crisis") at long intervals, and more or less acute, more or less relieved by "miotics," during which the redness of the limbus and of the episclera makes its appearance. The distinction between "simple" and "inflammatory" glaucoma can be, as Löhlein has said, only a clinical label; but we may add that a similar dissociation would be the negation of the passage, which is always possible, from one form to the other. Finally all of these eyeballs, except that of Rönne, showed hypertension.

It is therefore logical to conclude that the predominating characteristic of these examinations should be the presence of inflammatory phenomena, sometimes very acute. It is moreover not without interest to note that the leucocytic infiltration has its seat in the uvea, preferably in the anterior uvea, with very frequent propagation to the perforating episcleral vessels. There is no question either in these anatomic protocols, of a Knies closure of the angle characteristic of old glaucomas and absolute glaucomas. But we sometimes find mention made of a partial synechia, which is not surprising as inflammatory phenomena, even slight, are found also in the iris with remarkable constancy.

The case of Rönne, itself a type of simple chronic glaucoma without hypertension for several weeks, showed leucocytic islets in the superficial zone of the stroma. It must be remarked

also that except in this last case, the tissue of the iris was always the seat of a manifest rarefaction, this atrophy of the iris is therefore a very frequent phenomena. We may add that it may be discovered clinically by transillumination, that it is sometimes extremely marked[23]. In one of our patients it was so marked that it gave the impression of being an operative coloboma. Another fact, likewise constant, is that of pigment, either free or intracellular. When it is abundant, it is found everywhere, even in the vessels of the optic nerve. When it is scarce its presence is noted in the iris and at the level of the angle. We know, moreover, that in the living eye this pigment is very often perceptible with the slit lamp, and that Koeppe considered its presence in the iris as a sign of "preglaucoma." It is found again so regularly in anatomic sections that some have even thought that it was responsible for a "blockage" of the angle. Yet Hansen found it again in eyeballs taken from aged persons and from diabetic patients in whom no objective symptom had caused any thought of an affection of the eye. These pigmentary infiltrations, remarked as early as 1908 by Panas and Rochon-Duvigneaud in various inflammations, do not seem therefore to be pathognomonic of glaucoma. But, on the other hand, as they do not exist in the normal eyes of young people, we must in all logic suppose that they are due to a lesion in the cells which contain them. This pigment, not being of hematic origin (the ferric reactions are negative—Hansen), cannot but come from the epithelial cells of the pigment layer and from the chromatophores of the iris. Now, a cell suffers when it is illnourished or attacked by inflammation, and its contents then escape; and, as it is a visible substance in this case, we find this pigment either in free grains, or in cells which have phagocyted it, leucocytes and conjunctival macrophagi. But at the base of the phenomenon there is a vascular, obliterating lesion, or an inflammation; and both of these phenomena are to be found in glaucomatous eyes. Should we for that accuse the infiltration (taken in its most general sense) of causing the hypertension? Evidently not. We know as a matter of fact that many inflammations of the anterior segment are accompanied, on the contrary, by hypotony and that it is only accidentally that such and such an iridocyclitis or iridochoroiditis takes the opposite evolution.

To produce hypertonic phenomena it is necessary that the inflammation should affect the venous system, or else that the capillaries should dilate. It is the same in the limbs, in which a venous lesion alone is capable of engendering phlegmasia alba dolens. It would therefore be beginning at the wrong end to consider the hypertension of the eye as bringing about the inflammation, and Bjerrum[24] recalls very appropriately the experiment of Grönholm*. In the eye, in consequence of the experiments of Weber, of Schulten, of Koster, etc., on the hypertonic phenomena caused by ligature of the vortex veins, the attention of ophthalmologists was directed for a time towards the return blood channels of glaucomatous eyeballs. It is a remarkable fact, that all the protocols we have summed up mention venous lesions. In the majority of cases endo- or periphlebitis has invaded the vessels of the circle of Leber and the scleral perforating veins. In other cases the phlebitic process has reached as far as the vortex veins and even in the case of Rönne (chronic glaucoma without hypertension), the author states that the only vortex vein which could be discovered in the sections showed, at the level of the scleral crossing, a concentric stricture which reduced by one-third the diameter of the vessel. The anatomic examination of this case of simple chronic glaucoma had been singularly negative!

The reason is that inflammation is often a very fleeting phenomenon. Sometimes, after having been very acute it disappears; and we find only its traces. We have an example of this in infantile glaucoma, which is due to an intra-

*The experiment of Grönholm proves that we may, by increasing artificially the ocular tension, empty the vessels of their contents but not produce inflammation.

uterine inflammation, which at birth may have disappeared or still persist. When it has disappeared we find, none the less, arrests of development (such as milky cornea, as in the 4th and 5th months of gestation, microphakia) or monstrosities (aniridia, megalocornea) which may extend to the eyelids and to the face. When the morbid phenomena persist the hypertension which results from them, acting on the young tissues, distends them; and transforms the glaucoma of infancy into buphthalmia. In all the anatomic examinations of infantile glaucoma there exist phlebitic lesions, (Magitot[25]) which appear in the form of a more or less marked leucocytic inflammation, which tends toward fibrous degeneration. We must therefore not be astonished that nearly all the writers having had occasion to examine the eyes of children suffering from this malady, from Reis, Seefelder down to Meller, have drawn attention to the absence of Schlemm's canal.

The Schlemm canal is a part of the venous circle of Leber, its absence denotes an aplasia of the venous system, attacked by the inflammation in the course of its development.

The experimental results obtained by ligature of the vortex veins in an animal, evidently do not reproduce human glaucoma, but they possess a symptomatic value. The mistake is to always wish to find their pathologic obliteration in glaucoma, whether by the inflammatory process alone, or by a valve mechanism rendered possible by a congenital disposition (Heerfordt). The endophlebitic processes do not necessarily always extend to the large channels of exit. The phlebitis may have seated in the afferent branches, reach certain vortex veins, or be localized in the venous circle of Leber, whence it easily spreads to the perforating scleral veins which constitute the return anterior channels for the blood. Then stasis accompanied by hypertony will manifest itself, as after ligature of the vortex veins, which affects the extraocular channels instead of acting on the intrabulbar vessels.

We think it useful to recall what may be observed experimentally after ligature of the vortex veins. The phenomena have been perfectly studied by Koster[26] as results from counterexperiments made by one of us (Magitot).

In the rabbit, dog or cat ligature of the 4 vortex veins causes a severe hypertension which reaches a figure intermediary between the diastolic arterial pressure and the systolic arterial pressure without, however, ever reaching the figure of the latter.

In the rabbit, Koster says, after ligature, the anterior chamber becomes shallow, the cornea loses its sensitiveness, the pupil is large and round, the iris is congested and its base is pushed towards the cornea. The media remain transparent, which enables us to see the retinal veins distended and the papilla pink and slightly edematous. At the end of an hour, the sensitiveness of the cornea returns, partially, but chemosis appears, the lens begins to get cloudy, but we can still see that the retinal vessels are full of blood. Then the media become cloudy. Very frequently hyphema supervenes. The hypertension continues to remain about 65 mm. Hg.

At the end of a few days the cornea thickens and becomes milky and striated as in parenchymatous keratitis, then towards the limbus appears a slight vascularization, which increases rapidly then regresses. The cornea becomes transparent again. At the same time an intense vascularization shows itself in the ocular muscles, and towards the 5th day the anterior chamber clears up. It recovers its dimensions, but synechiae often persist. Lastly the fundus becomes visible with or without crystalline opacities. Generally the retinal arteries are filiform, the veins well filled, the papilla shows the glaucomatous cup. The tension remains very high up to the 6th or 7th day, then towards the 10th, it approaches normal. Koster, who had recourse in the animal sacrificed, to anatomic injections in the arteries, has recognized that this lowering of the tonus was concomitant with the formation of collateral emissaries, in place of

the vortex veins. He also saw that, almost without exception, a compensatory circulation was likewise visible in the sclera.

Partial ligature of 2 or 3 vortex veins. Results variable. In general the eye becomes hard, the iris is hyperemic, the ciliary body swollen, the pupil is oval and reacts slowly. The anterior chamber remains normal and the media transparent. Then the hyperemia of the iris soon disappears. As for the hypertension it remains high for several days, then it diminishes while remaining for a long time above the tension of the other eye. The lowering of this hypertony coincides with the appearance of compensating perforating scleral veins. In the dog the phenomena are very similar.

These experiments concern the vortex veins, that is to say, the posterior venous channels. It was interesting to know whether the suppression of the anterior channels of discharge was also capable of causing hypertensive phenomena.

The trial was made by Bartels[27] who tried to reach these channels by subconjunctival ligature of the 4 recti muscles. This operation set up in the rabbit and in the dog the same phenomena as the ligature of the vortex veins: conjunctiva injected, insensibility of the cornea, great hypertony which, in the dog, may persist for several weeks and even several months. The anterior chamber in the dog remains deep and there is no cup of the disc. *In the anatomic sections the veins remain empty of blood and the choroid does not appear to be distended.* The sole doubt that may be expressed is that the ligature, being blind, does not take in at the same time as the brush-shaped veins of the scleral edge, the vortex veins which come out under the upper and lower recti muscles.

The experimental obstruction of the return channels does not perhaps reproduce the human affection; but it brings about in any case a considerable hypertony and certain symptoms which singulary match those observed in acute glaucoma. Other effects are common to the acute and chronic form. Thus there supervenes after a longer or shorter time the appearance of a collateral circulation, which evokes the tortuousity of the episcleral vessels and the Medusa head. In the iris we note mydriasis, and in the retina we see the extreme repletion of the veins, flattened out at their angular exit on the papillary ledge. Finally in certain animals (the cat) the arteries show pulsation (Magitot[28]). We also draw attention to a remark made by Bartels in the anatomic examination of eyeballs with experimental hypertensioned. Never was this writer able to observe in dogs anterior veins gorged with blood, or even a distended choroid. This fact must be emphasized *for it answers an objection concerning the part of the blood stasis in human glaucoma,* a disease in which histologic protocols mention fairly often an absence of vascular repletion.

Such as they are, these experiments have value and they cannot be neglected in favor of attempts to provoke a retention of the aqueous humour. The attempts of Schreiber and Wengler, Erdmann, Parisotti, Scholer, Heisrath, et al. to block the region of the angle by cauterization of the limbus or electrolysis of the anterior chamber, have in no wise the demonstrative value attributed to them by their authors. The hypertension they set up is due to the destruction or the obliterating inflammation of the venous channels of the scleral circle, so highly developed in the mammalia, and is only another way of placing an obstacle in the way of the return circulation. The same remark applies to the hypertension set up by injection into the anterior chamber of the rabbit of nigrosin or isamin blue (pyrrohl blue) as Hamburger[29] very justly remarks.

THE CAPILLARY NETWORK AND THE NERVOUS ELEMENT.

The obstacle placed in the flow of the venous blood may be considered as the principal factor (principal does not mean sole) which creates a lasting hypertony with glaucomatous symptoms. When there is no obstacle but merely a hypertrophy of the vessels, an increase of the ocular tonus is indeed remarked but there are no glaucomatous phenomena. This is what happens sometimes in clinical practice. Elschnig has reported the case of a nevus of the face, (hemiface) in which the eyeball had a tonus of 36 mm. Hg. The conjunctiva was purple, the

retinal arteries as large as the veins. The whole eyeball had increased in volume without, however, any buphthalmos.

This example shows that a vascular malformation may bring about an exaggerated nourishment of the tissues and an unusual enlargement of the organ. The blood column not being hindered by any obstacle, the increase of the tension is but the result of the abnormal development of the capillary network. Per contra, when during intrauterine life an inflammation supervenes which, while causing a facial malformation, spreads to the veins of the eyeball, there is a hindrance of the circulation and we witness the evolution of infantile glaucoma as in the cases of Cabannes, Michelson-Rabinowitch, Coronat and Aurand.

Any inflammatory or noninflammatory obstacle to the return circulation may in fact cause a rise of the ocular tension. An example of this is furnished by the cases of arteriovenous orbital aneurisms. Bailliart and Poulard[30] have reported the observation of a man suffering from an arteriovenous aneurism of the orbit. In spite of ligature of the primary carotid the ocular pressure had remained distinctly higher on the side affected, and later on it was observed to rise as high as 40 mm. Morax recalled a similar case.

In this affection, the considerable distension of the veins of the eyelids and of the conjunctiva is a sign of profound disturbance in the venous circulation. Sometimes there supervenes an inflammatory element which spreads to the eyeball. We then observe distinctly glaucomatous phenomena as in the curious observation of Elsching.[31] The patient was a man of 47 who, in consequence of receiving a bullet from a revolver was suffering from exophthalmos. A few months later, rather suddenly the pulsations of the eye ceased, the subconjunctival veins suffered a thrombosis, the eyeball was red and hard. The pulsations had persisted in the upper eyelid in which the veins were much dilated. Later on the acute phenomena in the eyeball abated, the exophthalmos diminished, became pulsatile anew and Elsching observed the evolution of a chronic glaucoma.

In this observation it seems to be very evident that the spreading of an inflammation to the vortex and ciliary veins brought about acute glaucomatous phenomena, in an eyeball up till then unaffected. Elsching remarks likewise, that the symptoms abated parallel with the decrease of the phlebitis, but continued to evolve in a chronic manner. We may therefore logically admit that inflammatory lesions engender in the intraocular veins persistent disturbances, manifesting themselves objectively under the clinical form of chronic glaucoma.

Such examples concern cases of mild partial phlebitis of the orbital veins. We say partial, for total phlebitis, that which ends in inflammation of the sinuses, causes also a considerable exaggeration of the ophthalmotonus; but the rapidly mortal evolution of the disease usually distracts the attention toward the meningitic symptoms. We may also cite another category of inflammations of the orbit which have a remarkable action on the tension of the eyeball. They are the cases of metastatic cellulitis and of tenonitis which cause exophthalmia and chemosis. Then the ocular hypertension has a double mechanical origin; to the hindrance in the venous circulation is added the compression of the eyeball by the inflamed tissues which enclose it. When this compression is of some duration it may even engender in the retinal circulation the same phenomena as those observed in glaucoma.

Case of Orbital Cellulitis Complicated with Hypertension Choroiditis. Woman of 35, undergoing treatment for a pelviuterine affection, was suddenly attacked by exophthalmia, chemosis, loss of sight, edema of the eyelids and arching of the malar region. Hemocultures, Wassermann, bacteriologic examinations, negative. Pupil dilated with greenish gray reflection in the bottom. The hypertension of T = 55 mm. Hg. lasted a fortnight in spite of the miotics. Little by little the affection decreased, leaving behind it traces of a severe posterior choroiditis.[32]

Case of Tenonitis. Seefelder[33] reports an observation of tenonitis, accompanied by a violent attack of glaucoma which abated rapidly under the apparent influence of a salicylic treatment. One week later, there were tenonitis and a

glaucomatous attack in the other eye; but the phenomena were less severe and disappeared in 3 days.

These examples show the indisputable influence of the return circulation of the blood. In our opinion indeed the prime cause of all acute or chronic glaucoma is a vascular inflammation. *The fact that certain anatomic examinations of simple chronic glaucoma showed no trace of this does not signify that the inflammation has not existed at an early epoch of the disease.* This inflammation may have disorganized the venous system before disappearing, leaving behind it a process of sclerosis, difficult to appreciate in the sections. We may add that examinations of young cases are exceedingly rare, and the sole case here cited (Ronne's) concerned an eye of which the tonus had become normal again for several weeks after disappearance of the glaucomatous phenomena.

Lastly, if at the base of morbid phenomena a vascular lesion exists, it would be childish to deny the influence of a second factor, that of the *nervous element.* If indeed, from the experimental point of view, the best way to provoke a durable ocular hypertension is to act on the venous channels, we know that in an animal, a simple irritation of the iris (Magitot), a subconjunctival injection of a strong saline solution (Wessely), contusion (Leplat) is capable of causing an increase, sometimes considerable, of the ophthalmotonus. *Here again, experiment retains its full value, for clinical practice supplies us with similar cases.* We would recall to mind on this subject, the hypertony succeeding an operative prolapse of the iris, that which follows capsular extraction, and the striking observation of one of us concerning a patient suffering from luxation of the lens which, every time it fell over into the pupil, set up typical glaucomatous phenomena disappearing immediately after reduction by massage. And what better proof of the influence of the nerves than the hypertony (variable from one day, or one hour to another) which surpervenes in consequence of certain ocular contusions (Magitot[34]) and how can we explain otherwise the increase of the tonus and the choroidal hyperemia which may be observed in the eyes of animals undergoing experimentation (Leplat).

The influence of the nerves is likewise a very remarkable factor in the history of acute or chronic glaucoma and is sometimes so manifest that some, like Sulzer, wished to isolate a clinical type of "nervous glaucoma." But we must make ourselves clear! The fact that an attack of glaucoma supervenes in consequence of great emotion does not mean that the vasomotor element alone is concerned, and everything tends to prove that it is something similar to that played by the shock which causes a cartridge to explode. The ground must be suitable for the attack to start. This favorable condition results, we feel sure, from a relative hindrance of the return circulation, constituted by *a more or less marked diminution of the calibre of the veins,* the sclerosis remains of an endophlebitis, sometimes an old one.

In the normal state, the blood brought by the arteries, transmitted to the capillaries finds a sufficient outlet in the venous ramifications. But should a dilatation of the capillaries supervene, immediately the quantity of blood transmitted to the veins increases, the liquid column no longer finds sufficiently broad channels; the pressure tends to become equal in the veins and in the arteries, exceeding sometimes the diastolic figure (without ever exceeding the systolic low-water mark); and, no less immediate result, the ocular tension increases. If at this moment we inject into the orbit a solution of adrenalin, which contracts the posterior ciliary arteries, *we diminish the quantity of blood arriving* and the blood column falls. We modify therefore the circulatory regime (and at the same time the ophthalmotonus); and that may even *favor a return of the vascular constricting function,* tho this is unfortunately but a temporary activity, as is shown by the diagrams of Kadlicky.[35]

Thus the raising of the capillary dam by nervous vasodilating action may, in certain favorable cases, provoke a local hypertensive attack. Does not emotion cause the face to grow pale and then flush, i. e. contract and dilate the cutaneous capillaries? But on other occasions, the nervous element does not participate in the disturbance of the circulatory regime. The capillary dilatation is then *pas-*

sive. This is what takes place *when the general arterial pressure increases suddenly,* as in angiosclerotic patients after a heavy meal. Then the ocular capillaries allow themselves to be distended without reacting, they transmit to the venous network a quantity of blood, which cannot flow off freely, and the attack of hypertony commences.

Here again clinical practice agrees with the results of experimental research. We may in fact cause a general arterial hypertension by injecting into the femoral vein of a dog a certain quantity of adrenalin or pituitrin. These drugs act by provoking a spasm of all the peripheric small arteries. The point of attack is therefore the same as the hypertension in angiosclerosis where the increase of the blood pressure results in a diminution of the calibre of the peripheric vessels, an affection which is often accompanied by a spasm.

Now, experience shows (Henderson and Starling) that after injection of adrenalin into the circulation, the ocular capillaries may either take part in the vasoconstriction, or let themselves be forced by the blood column. In order that they may let themselves be forced completely, it is sufficient to set up a slight local vasodilatation by subconjunctival injections of a salt solution as in the following. Magitot.[36]

Experiment. Chloralosed cat. Arterial pressure taken at the abdominal aorta. Ocular manometer in connection with the anterior chamber. Arterial pressure 90/100. Ocular tension 20 mm. A subconjunctival injection of 1 c.c. NaCl 15% solution, is made, the effect of which is to raise the ocular tension to 44 mm. Hg. A quarter of an hour after, a dose of 5 centigrammes of pituitrin (Choay) is injected into the femoral vein. The aortic pressure indicates a fall, then a sudden rise and reaches 210 mm. Hg. The ocular tension follows, but a little more slowly, the movements of falling and rising. It reaches the high figure of 125 mm. Hg. Thus, a simple local vasodilatation due to the previous subconjunctival injection of NaCl enables the arterial pressure to force the barrier of the ocular capillaries, and to cause the ophthalmotonus to rise proportionately to its own strength. We have thus the proof that *the increase of the general arterial pressure can force the dam formed by the ocular capillaries between the arterial and venous systems, and should any obstacle to the return circulation exist, the blood pressure tends to become equal and to approach the highest figure, that of the systolic arterial pressure.* It plays in this case an active but accidental part, capable of causing a hypertony in a system until then barely in equilibrium. *But most often, the general arterial pressure has not so much importance.* The eye feels its influence passively whether it be healthy or glaucomatous. But when it is glaucomatous, i. e. when it shows blood stasis, the modifications of this arterial pressure are singularly amplified, and this is comprehensive since the capillaries no longer play their regulating part and the whole vascular system of the eye tends to become a closed system. We then note in the same day, and from one day to another variations of the ophthalmotonus which may reach 15 to 20 mm. Hg. whereas in the healthy eye of the same patient the parallel differences of level do not exceed 5 mm. The diagrams published on this subject by Kollner[37] are particularly suggestive.

EFFECT OF THE OCULAR HYPERTENSION ON THE RETINAL CIRCULATION.

We may therefore suppose that in a glaucomatous eye, the pressure of the blood column in the capillaries of the uvea will be higher in proportion as the venous obstacle situated downstream is more important. The increase of the ocular tonus which will result will inevitably have its counter effect on the retinal circulation which is an autonomous system and totally independent of the ciliary vessels. Thus the increase of the retinal pressure will be but the inevitable consequence of the rise in the ocular tension. This effect is secondary, purely mechanical, and observable thru the ophthalmoscope. The retinal circulation is therefore able to supply us from a clinical point of view with useful information.

If the ocular tension rises above its normal figure, without reaching the diastolic pressure of the central artery, nothing in the examination of the retinal

vessels shows the circulatory disturbance. It exists, however, for the normal capillary pressure has been exceeded. The circulation in these small vessels remains nevertheless assured, because the crushing of the vein against the disc's border has raised the pressure in the whole system upstream. But it is a precarious regime, unfavorable to the good nourishment of the retinal cells which suffer and will end by atrophying. This atrophy begins in the retinal periphery, in the extreme arterial ramification. There follows that constant vascularization in the retina of eyeballs suffering from hypertension for some time, which in chronic cases spreads to the posterior pole. Often, in the beginning arterial spasms aggravate this disturbance and attacks of obnubilation supervene. But if the pressure increases and *reaches the minimum arterial pressure, pulsations appear.* We may conjecture that at this moment circulation is extremely precarious; the blood wave cannot make its complete circuit. The central vein crushed against the papillary disc is impervious, hence edema, which is accompanied by cloudiness of the vitreous humor, of choroidal origin.

Thus the danger appears chiefly at the moment when the ocular tension is equal to the diastolic pressure. We see therefore of what clinical interest it is to compare the ocular tension and the arterial pressure in all cases of glaucoma. For those who do not know to measure this local pressure and in the case in which the disturbances of the media prevents this being taken, we would recall that the *arterial retinal pressure is practically half the brachial pressure* which can so easily be measured by means of the armlet. In accordance with this, if *the ocular tension equals, or still more so, if it exceeds half the minimum brachial arterial pressure, there is reason to be alarmed*; and if miotics do not restore the normal equilibrium, recourse must be had to operation. In cases in which considering the fairly good state of vision, we hesitate sometimes as to the urgency of the operation, a rule thus formulated, based on experience, should be remembered.

The rise of the ocular tension may go still further; *it may approach the local systolic pressure* (*blindness is then total*), *but it can never go beyond it.* Let us imagine indeed an ocular tension stronger than maximum local arterial pressure; it means a complete stoppage of the circulation and consequently the death, no longer simply functional, but also anatomic of the eye. The few cases reported, of bursting of the eyeball in complete glaucoma, might perhaps be explained by the disorganization of the ocular walls under the prolonged influence of an ocular tension closely approaching the systolic pressure. The ocular hypertony being the result of an abnormal dynamism of the intraocular liquids (circulating or noncirculating) it is difficult to conceive this pressure capable of being superior to the arterial pressure when this arterial pressure alone can create it. If we wish to make certain of this maximum arterial pressure, often so difficult to measure, we may say that it is practically 60% of the maximum pressure taken in the arm.

On all these points, clinical practice is once more in agreement with the experimental practice. Here is the proof:

Experiment: (Magitot). Chloralosed cat. Aortic pressure of 130, 140 mm. Hg. The vertical veins are tied. The ocular tension raises to 80 mm. and settles down 70 mm.

The ophthalmoscopic examination enables us to note that if the retinal veins are full and motionless the arteries are, per contra, animated by pulsations. A very slight pressure (20 grammes) on the eyeball suffices to drive the blood out of the arteries.

Conclusion: The ocular tension being 70 mm. Hg., the systolic pressure in the central artery must be very near this figure.

REMARKS AND CONCLUSIONS.

The reader will remark that we have up to the present avoided speaking of the aqueous humor. Our purpose is not in fact to open like Hamburger and Seidel a discussion for or against the theory of filtration. We merely wished to show that the hypothesis of glaucoma as a nervous disease is not a reasoning ad absurdum but it may be supported by numerous facts. This being said, we would draw attention to a few particular points.

First of all, in many glaucoma cases, the aqueous humor is not increased in the volumetric sense. On the contrary, it may be certified that the greater the tension, the less is the quantity of humor furnished by puncture of the anterior chamber. This observation agrees with the protrusion of the iris, or of the ciliary process, the swelling reported by Schmidt-Rimpler[38] (Graefe-Saemisch) and seen directly by Hess[39] by means of his apparatus. Only the glaucoma of children may have great hypertension with a deep anterior chamber, which is accompanied moreover by an increase in the volume of the eyeball. The same thing takes place in animals of which the hypertended eye allows itself to be distended.

Another curious fact which shows how chemically indifferent the aqueous humor remains to the modifications of the tonus, is that in an animal, in spite of ligature of the vortex veins having set up a hypertony of more than 70 mm. Hg., puncture of the anterior chamber followed by analysis with repuncture effected half an hour after the operation shows that the composition of the liquid has not changed. (Magitot and Dautrevaux).

But this is not all, clinical practice once more justifies experimental practice. It is indeed well known now that, in chronic glaucoma without inflammation, the composition of the aqueous humor is almost normal (Ascher, Magitot and Dautrevaux). There is chemical modification only if there is inflammation. Then the albumin increases, the chlorides diminish, the formula of the aqueous humor approaches that of the blood serum.

We do not insist, but it appears strange that secretory modifications are not accompanied by modifications in the composition of the liquid. As to the inflammation, it always provokes (whatever its cause may be), an increase in the proteid substances; as in keratitis, iritis, etc.

Finally, here is another fact which is constantly observed: A glaucomatous eyeball under great tension is enucleated. After the operation the tension goes down, but it remains for sometime harder than a normal eye; a fact reported formerly by Priestley Smith. Why? Because of the obstruction of the angle? This hypothesis is useless. It has been proved that a healthy eye possessing its own pressure of 20 mm. retains for several hours after enucleation a tension of 10 mm. Hg. and does not become truly dead until 15 hours after. The difference between 10 mm. Hg. the figure after death, and 20 mm. Hg. the figure during life, is due to the blood pressure. We are therefore justified in considering the aqueous humor as a dialysate (Magitot and Mestrezat, de Haan, Van Crefeld, etc.), which has been stored up during life under a certain osmotic pressure and which instead of escaping by the angle of discharge remains in the eyeball enucleated or deprived of blood until the cells which border the space bathed by the liquid die in their turn. The cellular membranes become pervious only at the moment when they begin to decay. The proof of this is that if we keep the eyeball immersed in hemolysed serum in a refrigerator this moment is delayed for several days[40]. The same thing occurs in an eyeball under hypertension. Should we try by this special process to preserve a glaucomatous eyeball with a view to making use of it for grafts of the cornea, we may observe that at the end of several days this eyeball still possesses a tonus slightly superior to that of an eyeball without hypertension preserved in the same way[41].

It must be added that apart from all question of preservation, if we compare two enucleated eyes, one normal, having a tonus of 20 mm. Hg., the other with hypertension of 50 mm. Hg. before operation, it will be remarked that if the tension of the former falls to 10 mm., the tension of the latter will fall considerably more, to about 20 mm., which by contrast will still seem very high. However it may be, we may admit that the difference from 10 to 20 in the first and from 50 to 20 in the second is due to the blood column. What remains of the tension belongs to the aqueous humor which has been stored up under a certain osmotic pressure and under a certain hydrostatic

pressure (blood pressure). The osmotic pressure which is related on the one hand to the composition of the blood and on the other to the porousness of the cellular membrane, appears to be but very slightly modified in glaucomatous states. On the other hand the hydrostatic pressure of the capillaries is very high, as we have endeavored to prove.

In this paper we have indeed recalled that if the ocular tension is strongly influenced by the general pressure, it is above all regulated by the local circulation. In persons suffering from hypertension a low ocular tension, as happens sometimes, is attributable to the shrinking which affects the peripheric small arteries in general and the ocular small arteries in particular, whereas the supernormal ocular tension (the most frequent) is in proportion to the distension of these same vessels.

On the other hand, the observations show that there is normally a similarity between the figures of the blood pressure in the choroidal and in the retinal systems. The diastolic pressure is 30 mm. Hg., the systolic 70 mm. The venous pressure is on leaving the eyeball in equilibrium with the ocular tension. Such are the conditions in a healthy eye.

In glaucomatous hypertensions these conditions are profoundly modified and the vascular regime is characterized by a considerable elevation of the venous pressure, both in the choroidal and the retinal systems. But the anatomic separation being complete, between the two vascular systems, we cannot, in seeking the causes of this venous hypertension, merely draw conclusions from one to the other.

As concerns the choroidal circulation, anatomic and clinical study authorizes us to look upon the venous hypertension as being produced by phlebitic lesions, chiefly in the veins of the ciliary circle. These lesions may be more or less intense. They may evolve towards sclerosis while causing a reduction in the calibre of the return channels of the blood.

Basing ourselves always on experiment or clinical practice, we have likewise shown that the rise in the ocular tension of glaucoma might be attributed to this circulatory hindrance in the venous current. But we must consider the part played by the obstacle at the exit of the blood only in its effect on the capillary network. The capillary network normally forms a restraining barrier, which absorbs the arterial blood column and transforms its strong pressure into a weak venous pressure, but on condition that the flow of blood be not slackened. When old or recent venous lesions exist, equilibrium may still be maintained, if the obstacle is not too great. Should, however, an active vasodilatation supervene owing to nervous influence, the capillary network distends and ocular hypertension is produced. In the same way a sudden rush due to the increase of the general arterial pressure may likewise force the barrier of the capillaries and provoke the same phenomena. The inconstancy of the vascular dilation reported in the anatomic examinations of glaucomatous eyeballs does not gainsay this mechanism for the same inconstancy exists in the eyeballs of dogs rendered strongly hypertensive by venous ligature.

If we consider now the retinal circulation we may conclude that it undergoes passively the modifications of the ophthalmotonus. The reason for the rise in the pressure in the central vein must also be sought for in an obstacle to the outflow of the blood. This obstacle is due to the compression of the bent trunk of the large branches which, on the rigid plane of the disc, are more or less flattened by the pressure exerted by the interior media of the eye on their wall. There results a stasis with dilatation and repletion of the veins, which may be observed thru the ophthalmoscope. This stasis cannot, however, exceed a certain degree; for the same pressure which acts on the vein manifests itself likewise on the artery, and thus moderates the arrival of the blood.

When the ocular hypertension reaches the figure of the arterial diastolic pressure pulsations appear at the entrance of the central artery, because equilibrium being realized on both

faces of the arterial wall, the free play of this elastic wall becomes possible.

When the pressure of the ocular media exceeds the diastolic pressure, the blood can no longer penetrate into the artery, except at the moment of systole, and we may lay down the rule that if the ocular tension can sometimes approach the figure of the systolic pressure, it never, however, exceeds it under penalty of emptying the arteries of their contents.

From the practical point of view, the examination of the retinal circulation is capable of furnishing a basis for operative therapeutics. Danger appears in fact when the ocular tension equals, and still more so when it exceeds, the diastolic pressure. This datum is easily obtained, we merely need to remember that the pressure in the central artery of the retina is, in a healthy eye, practically half the pressure in the brachial artery.

BIBLIOGRAPHY.

1. Charron. Lille Thesis, 1922.
2. Wessely. Arch. f. Augenh., v. LXXXIII, p. 99.
3. Bailliart. Jour. Coeur. et Vaisseaux, Oct., 1920.
4. Bull. de la Soc. d'Opht. de Paris, 1922.
5. Weiss. Pflüger's Arch. f. g. Phys., 1906.
6. Fromaget. Ann. d'Ocul., 1921.
7. Wessely. Arch. f. Augenh., 1908.
8. Leplat. Ann. d'Ocul., 1924, v. CLXI, p. 87.
9. Magitot and Bailliart. Ann. d'Ocul., 1921.
10. Graefe's Arch. f. Ophth., v. XXXI and XXXII.
11. Stirling. Royal London Ophth. Rep., 1893, v. XIII.
12. Roser. Arch. f. phys. Heilkunde, 1859.
13. Schoen. Graefe's Arch. f. Ophth., v. XXXI.
14. Koster. Graefe's Arch. f. Ophth., v. XXXIX.
15. Heerfordt. Graefe's Arch. f. Ophth., v. LXIII.
16. Birnbacher and Czermak. Graefe's Arch. f. Ophth., v. XXXII.
17. Birnbacher. Fest. d. k. k. Univ. z. Gratz, 1890.
18. Levinsohn. Arch. f. Augenh., v. LXII, p. 131.
19. Hussel. Zeit. f. Augenh., v. XXVII, p. 213.
20. Thomsen. Klin. M. f. Augenh., v. LX, p. 339.
21. Hanssen. Klin. M. f. Augenh., v. LXI, p. 609.
22. Rönne. Klin. M. f. Augenh., v. LI.
23. Bergler. Arch. f. Augenh., v. XCV, p. 35.
24. Bjerrum. Klin. M. f. Augenh., v. L, p. 42.
25. Magitot. Ann. d'Ocul., 1912.
26. Koster. Graefe's Arch. f. Ophth., v. XLI.
27. Bartels. Zeit. f. Augenh., 1905, p. 97.
28. Magitot. Ann. d'Ocul., 1923.
29. Hamburger. Klin. M. f. Augenh., 1922, p. 69.
30. Bailliart and Poulard. Bull. de la Soc. d'Opht. de Paris, 1921, p. 21.
31. Elschnig. Graefe's Arch. f. Ophth., v. XCII, p. 101.
32. Magitot. Bull. de la Soc. d'Opht. de Paris, 1924.
33. Seefelder. Wien. med. Woch., v. LXXIV, No. 40.
34. Magitot. Ann. d'Ocul., 1917-1918.
35. Kadlicky. Arch. d'Opht., 1924, p. 704.
36. Magitot. Ann. d'Ocul., Feb., 1923.
37. Köllner. Arch. f. Augenh., 1920, v. LXXXVI.
38. Schmidt-Rimpler. Graefe-Saemisch Handb.
39. Hess. Arch. f. Augenh., v. LXXXIV.
40. Magitot. Ann. d'Ocul., Feb., 1923.
41. Magitot. London Ophth. Congress, 1913.

MUSTARD GAS BURN OF THE CORNEA.

DONALD J. LYLE, M. D.

CINCINNATI, OHIO.

A man exposed to mustard gas in action suffered burns of the face and severe conjunctivitis. The corneas became steamy followed by opacity of the left. Four years afterwards there was photophobia, conjunctival hyperemia, pannus and epiphora.

In my association with the examining staff of the Veterans' Bureau for about two years I found only one case that showed permanent ocular lesions from mustard gas burns received in the World War. The case is that of a man, aged 26, whose examination report, upon entering service thru a

Fig. 1.—Gas burn of cornea, photophobia.

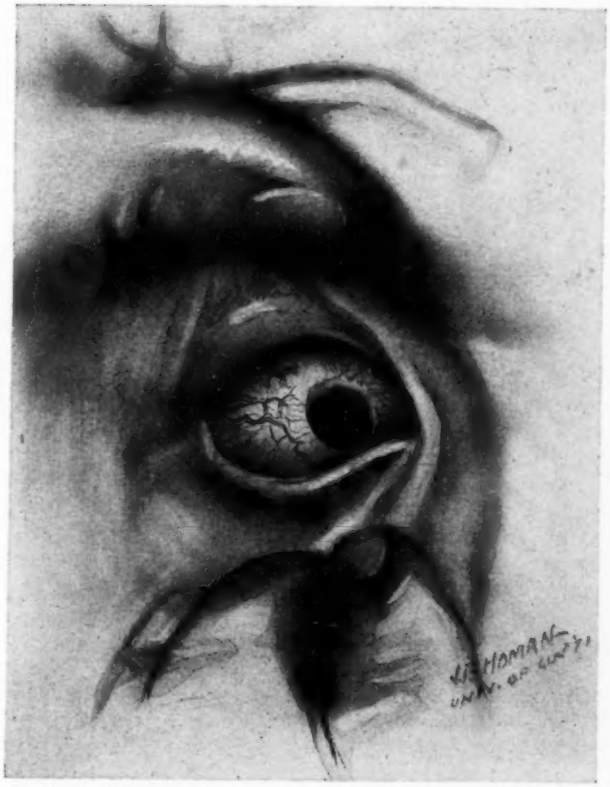

Fig. 2.—Gas burn of cornea, right eye. Conjunctival hyperemia, corneal opacity.

local draft board, gave his vision as 20/20 both eyes; no eye defects were noted. An abstract of his medical service record shows the following:

Sept. 19, 1918. Gassed—mustard. Action. Contact—face, eyes, right forearm and hand. Severe. Inhalation.

Sept. 29, 1918. Diagnosis confirmed. Gas Evacuation Hospital No. 3.

Oct. 7, 1918. Complains of face, hands and eyes burning and sore chest.

Oct. 20, 1918. Eyes much improved. Right, 20/30; left, 20/100.

Nov. 1, 1918. Conjunctivitis still present—burns all healed—lungs clear.

November 25, 1918. Skin on face and neck healed.

Nov. 30, 1918. Silver proteinate twice daily.

Dec. 5, 1918. Eyes now only photophobia. Conjunctiva still edematous.

Dec. 6, 1918. Received at Base Hosp. 22. Left eye opacity of cornea upper

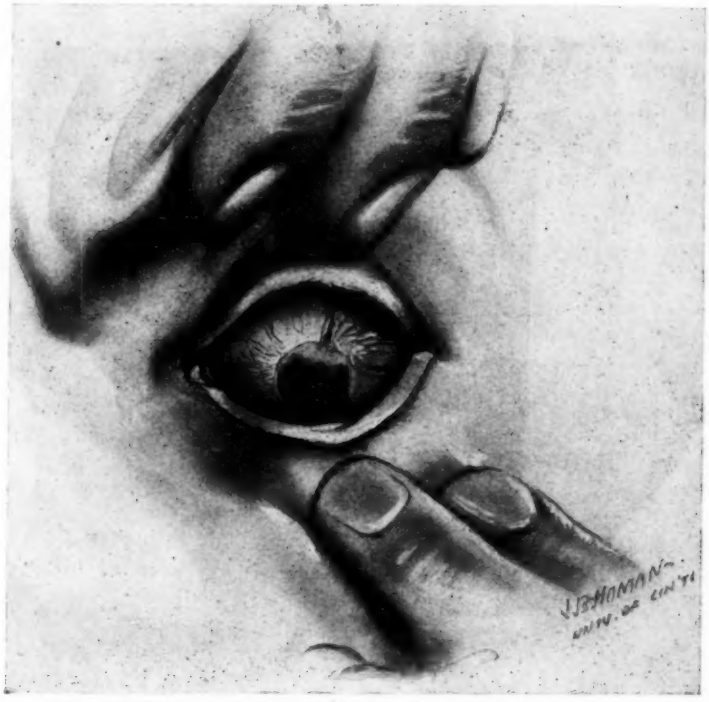

Fig. 3.—Gas burn of cornea left eye. Scar and hyperemia above.

Severe conjunctivitis. Second degree burns of face, neck and scalp. Moderate inhalation condition—few râles in lungs—heart O. K. All due to mustard gas.

Oct. 8, 1918. Conjunctiva much congested, edematous, cornea some steamy, pupils small. Treatment—Alkaline wash, Silvol 20%, Albolin. Steam inhalation with menthol, Amm. Cl. gr. x Sod Bicarb. t.i.d., Chloratone spray, Atropin. Right 20/40. Left 20/50.

Oct. 10, 1918, cornea steamy, ocular conjunctivitis—some edema. Alkaline wash, albolin, vaselin to lids.

quadrant. Left, 20/50; right, 20/20. Bilateral thickening of ocular conjunctiva.

Dec. 11, 1918. Smear—Hay Bacillus.

Dec. 20, 1918. Smear—Morax Bacillus.

Dec. 22, 1918. Conjunctivitis much improved. Petrolatum lig. Argyrol 20% t.i.d.

At the time I first saw the man, four and a half years after the injury, his brow was furrowed, with his eyelids drawn almost together as tho he were looking at an intense light. His chief

complaints were pain in front of the eyes especially the left: photophobia: blurred vision.

The globes and orbits appeared normal. The lids, held nearly shut, formed a palpebral slit: the brows were furrowed. Conjunctivae were markedly injected: the scleral and episcleral vessels were quite evident. There was no sign of scar tissue or granulation in the palpebral conjunctiva. The right cornea had a pannus on its superior nasal margin, extending inward toward the pupil about 2 or 3 mm. The pannus of the left cornea was mostly in the upper third, with extension along the nasal and temporal margins. Blood vessels were seen in the pannus of both eyes. The lacrimal system seemed to function properly, there was epiphora. Tension was normal. The pupils were equal, round and normal in size, reacting directly and consensually. The field of vision was normal except for a slight constriction due to pannus in the left eye. Fundi were negative.

Vision, right eye was 20/70, corrected to 20/20 with cylinder minus 1.50 axis 90; left eye vision 20/100 corrected to 20/40 with sphere minus 2.00 combined with cylinder plus 3.00 axis 150.

There was no history of prewar burns, injuries or diseases of the eyes. The evidence is sufficient to indicate these corneal lesions are the result of mustard gas burns with, or possibly without subsequent infection.
19 W. 7th St.

TUMOR OF THE ORBIT, OF DOUBTFUL ORIGIN.

MAJOR R. E. WRIGHT, I. M. S.

MADRAS, INDIA.

This case was seen at the Government Ophthalmic Hospital, Madras. The tumor extended up from the orbit encroaching upon the anterior fossa of the cranium and was probably congenital. The prominent cell elements were, eosinophiles, phagocytes and endothelial cells.

L., Hindu, female, aged 8, was brought to the hospital for a growth above the right eye which had been there since birth. There was an ovoid swelling in the superior temporal region of the orbit. Its long axis lay obliquely, the lower extremity being about the level of the canthal line, the upper extremity being just above the eyebrow at the junction of its outer and middle thirds. It was about as prominent as the convexity of a tea spoon.

The upper lid was pushed down-

Fig. 1.—X-ray frontal showing bony cavity at superior temporal quadrant of orbit, outlined by dots.

wards and inwards so that even when the patient made an extreme effort to open the eye only a slit like palpebral aperture was apparent. On elevating the lid the cornea and conjunctiva appeared perfectly normal. There was no evidence of inflammation or pulsation. The swelling fluctuated, but did not give the impression of fluid; it did not disappear on pressure or cause any dis-

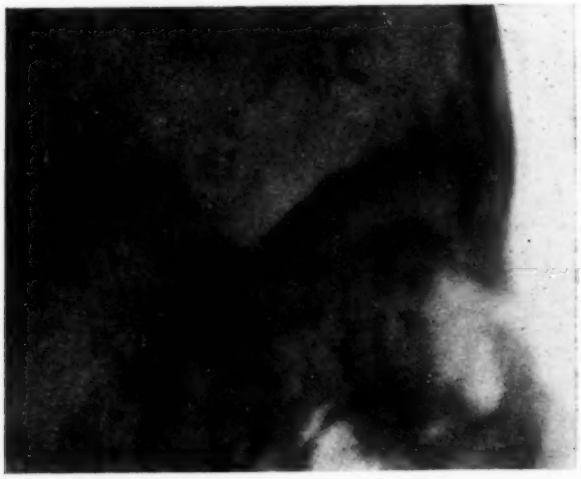

Fig. 2.—X-ray lateral showing bony cavity above orbit, outlined by dots.

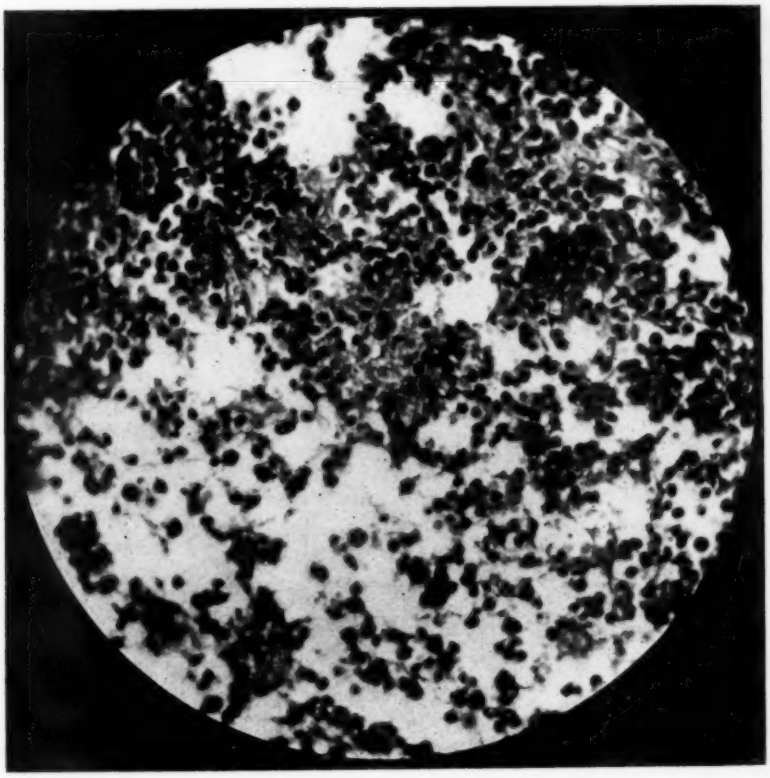

Fig. 3.—Structure of tumor showing endothelial cells, open reticulum and giant cells.

comfort, or evidence of cerebral disturbance. The finger could not be passed between the tumor and the orbital rim, but the edge of the latter was felt to be sharp and irregular and deeply notched. The skin moved freely over it and it was not attached to the globe nor was the latter displaced. It was considered to be a cystic dermoid (which is not an uncommon condition here), and the case was posted for operation, without a preliminary X-ray examination.

On cutting down over the mass it was observed that there was no definite capsule to be found. Apparently the wall of the cyst merged gradually into the surrounding tissues so that one could not find a line of cleavage. At this stage exploration with a large bore needle was carried out, and altho palpation indicated fluid, nothing was aspirated except some shreds of yellow amorphous material suggesting slough. The cyst was then carefully opened and to use the expression of my assistant a material "very like brain" presented. It was uncomfortably like brain, but like the brain that one sees at an area of softening, with a yellow tinge and with obvious necrosis. The soft boggy mass appeared to be definitely connected with the wall; it was confluent towards the center and on passing the finger into the opening, friable tissue separated and came out alongside the examining finger.

It was determined to explore further, and eventually it was found that the mass extended up above the level of the orbital roof toward the anterior cranial fossa for about three-fourths of an inch. An irregularly domed cavity about the size of a walnut was located in the superior temporal quadrant of the orbital roof. Having satisfied mysef that there was no obvious cerebral connection, or large area of dura exposed, the greater portion of the contents was removed with gauze and the cavity was loosely packed and drained. It granulated up from the

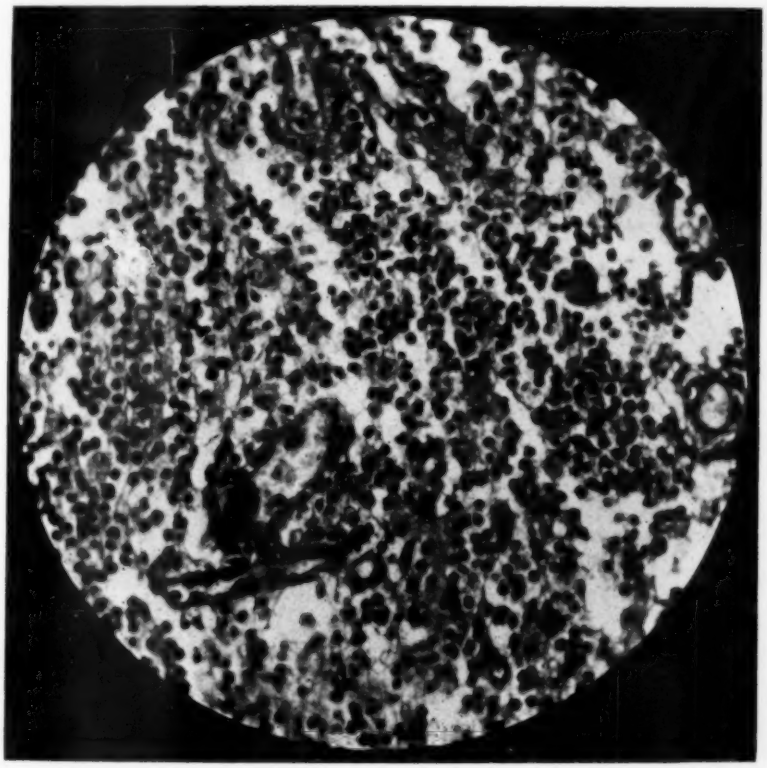

Fig. 4.—Section of tumor showing small vessels dead giant cells and massing of leucocytes.

bottom very quickly and within a fortnight there was hardly anything to be seen except a slight ptosis.

It should be noted that the lacrimal gland seemed normal in size and position, and free from the growth. An X-ray photograph taken after operation showed an irregular bony cavity in the superior temporal quadrant of the orbit encroaching on the anterior cerebral fossa. In anteroposterior extent it was about two-thirds of the depth of the orbit. In height it was about half the vertical height of the orbit above the normal roof level. Mesially it extended about one-fourth of the diameter of the orbital rim. The X-ray prints show this, but are not very sharp. The outlines of the bony cavity are dotted in for the sake of clearness.

THE HISTOLOGY of the tissue was very interesting. There was no real capsule nor anything that could be called a lining layer of cells. Quite different appearances were observable on the slide as one moved it about under the microscope, but the main impression was as follows: The ground is composed of endothelial cells with a very open intervening reticulum, without continuity in parts. These cells are in many places continuous with the walls of the smaller vessels which are composed of several layers of endothelium. There are evidences of multiplication of neuclei in these cells and here and there small giant cells are seen. In other places there are syncytial like aggregations.

Isolated large rounded phagocytic cells are scattered thruout the section, containing red blood corpuscles, eosinophiles, leucocytes and disintegrating nuclei. Many of these large phagocytes are dead and show the ghosts of cellular contents. There are large areas of leucocytic aggregation, and the cells are practically all of the coarsely granular eosinophile type with bilobed nuclei. Leucocytes and nucleated red cells are infrequent. Here and there this almost confluent cellular structure has undergone necrosis and there is little except debris of nuclear fragments taking the stain. With Leishman's stain the most prominent feature is the enormous number of eosinophiles, both in and around the capillaries thruout the reticulum, and forming large aggregations elsewhere. The large phagocytes too are well seen with this method, stuffed full of cells. No microorganisms can be found. The microphotographs for which I have to thank Director of the King Institute of Preventive Medicine, and Mr. Hawley of the staff, give a partial impression of the histologic structure.

HYDROPHTHALMUS.

H. D. LAMB, M.D.

ST. LOUIS, MO.

This is an account of the pathologic anatomy of an eye that had become inflamed and painful seeming to impair the child's general health. A report of the case by Dr. John Green, Jr., is included. The sections were prepared in Eye Research Bureau of the Missouri Association for the Blind and St. Louis University School of Medicine. Read before the St. Louis Ophthalmic Society, February 27, 1925.

Hydrophthalmus is congenital, primary glaucoma, occuring in infancy. In English speaking countries, such cases are commonly designated as "buphthalmus." It would seem better however to confine the term buphthalmus to all cases in which the eyeball is larger than normal. The Germans restrict the term "hydrophthalmus" to congenital primary glaucoma of infancy, which is accurately descriptive of the actual condition present.

As to the frequency of hydrophthalmus, we can report that out of 507 pupils in the Missouri School for the Blind during the last 20 years, 28 (5.3%) had become blind from this cause. Of this number, 20 were boys and 8 girls (males 71%). Strange to say this preponderance of males was found by Parsons, Gros, Zahn, Kunsmann and Seefelder. These authors report a total of 388 patients with hydro-

phthalmus, of which 63% were of the masculine sex.

The cause of hydrophthalmus is a fault in the development of the filtration angle, the latter remaining at an embryonic stage. Interference with development occurs in varying degrees at different points and is often not present thruout the whole circumference of the angle of the anterior chamber, in the same eye. In 1899 Polya found that the iris angle was stated to have been open in 47% of the recorded cases.

A difference of opinion prevails as to what precisely are the changes causing the blocking of the aqueous at the iris angle. Collins, Cross and others, conclude that this obstruction is due to an incomplete separation of the iris from the cornea; in other words that there persists a prehuman, mammalian, or prenatal condition of the ligamentum pectinatum. On the other hand Reis, Roemer and others think that the blocking is due to an aplasia, or lack of development of the venous network of Schlemm.

Collins and Cross regard the lack of filtration spaces at the angle as a secondary change following incomplete separation of the iris from the cornea and point to the secondary partial or complete obliteration of the canal of Schlemm in the glaucoma of adults as confirmatory of this view. It would seem difficult to explain how a few strands of tissue extending between the iris and cornea, as remnants of the prenatal or mammalian stage in the development of the ligamentum pectinatum, could cause such a complete blocking to the exit of the aqueous. This is particularly hard to understand when we consider that in the eye of mammals, with many strands of such tissue between the iris and cornea, glaucoma is very rare. Aqueous must be able to pass freely thru the openings between such strands, just as in the normal adult ligamentum, it flows thru spaces between the trabeculae.

CASE REPORT BY DOCTOR JOHN GREEN, JR.

D. J. R., male, was first seen June, 1918, when one day old. Both eyes were markedly hydrophthalmic. Cor-

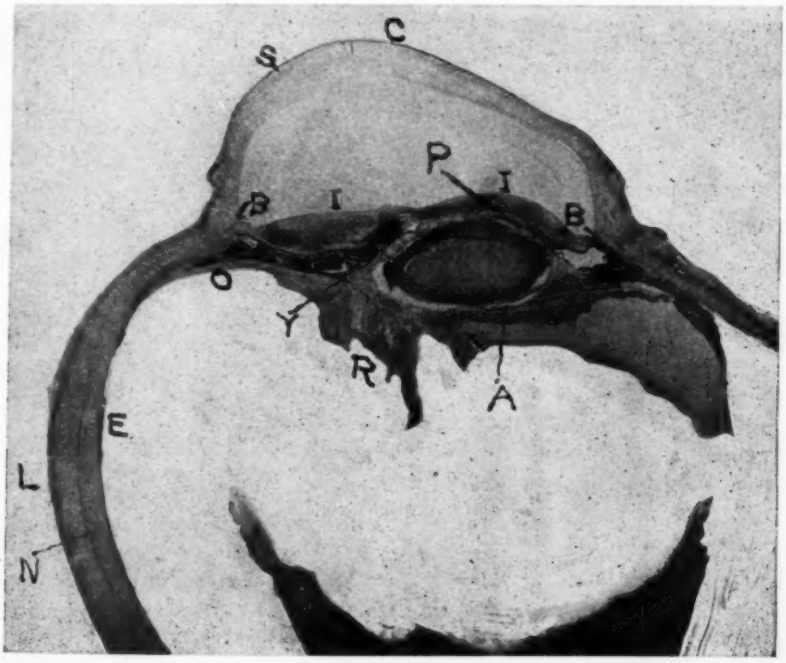

Fig. 1.—Section thru anterior portion of hydrophthalmic eye, showing C, thickened cornea with S, thin strip of cicatricial tissue, B, base of iris adherent to cornea, I, iris thickened with old hemorrhage, Y, cystic space between posterior epithelial layers of iris, P, posterior synechia, A, cyclitic membrane, O, atrophic ciliary body, R, completely detached retina, E, extensive proliferation from pigmented retinal epithelium, L, thickened sclera, with N, thin linear interspace derived from uveal tract.

neae were very large and diffusedly clouded. Tension (fingers) was much increased. Pilocarpin ½% and dionin 1% were prescribed for home use. In September, 1918, the tonometer (Schiötz) read R., 51; L., 71. At this time the patient was seen in consultation by Doctor A. E. Ewing, who counselled against operation at the time, but suggested sclerocorneal trephining later.

As the patient did not improve under miotics, each anterior chamber was tapped in June, 1919. There was some improvement; the cornea became clearer and the mother assured me that the child could recognize large objects. Paracentesis was repeated several times, each time with temporary benefit. In August, 1921, the patient recognized the position of a flash light held at 3 feet and walked directly to a flash light when held 15 feet away.

November 8, 1921, sclerocorneal trephining (Elliot) was done on the left eye. The very thin cornea was split with difficulty. Trephine rotated only a few times when the chamber was penetrated. The attempt to excise the iris at the same time as the trephine button failed, so the flap was replaced and sutured. A deep sulcus appeared in the sclera adjacent to the cornea. There was extreme hypotonus following operation.

March 21, 1921, patient appeared with a very acute conjunctivitis, both eyes. Treatment and examination very unsatisfactory, owing to struggles of patient. March 25, he was put under gas when it was found that the right cornea (this eye had not been trephined) showed a large central ulcer with deep infiltration. While examining the eye, the ulcer perforated, accompanied by a jet of aqueous. The dressings were immediately torn off by the patient. The right eye finally recovered with a flattened leucomatous cornea and moderate shrinkage.

The patient was not seen for over two years. He was brought again May 29, 1924, because the left eye "looked queer," when it was found that the anterior chamber was half filled with blood. (Father states that the child bumped the eye.) The eye was very soft. During the summer the

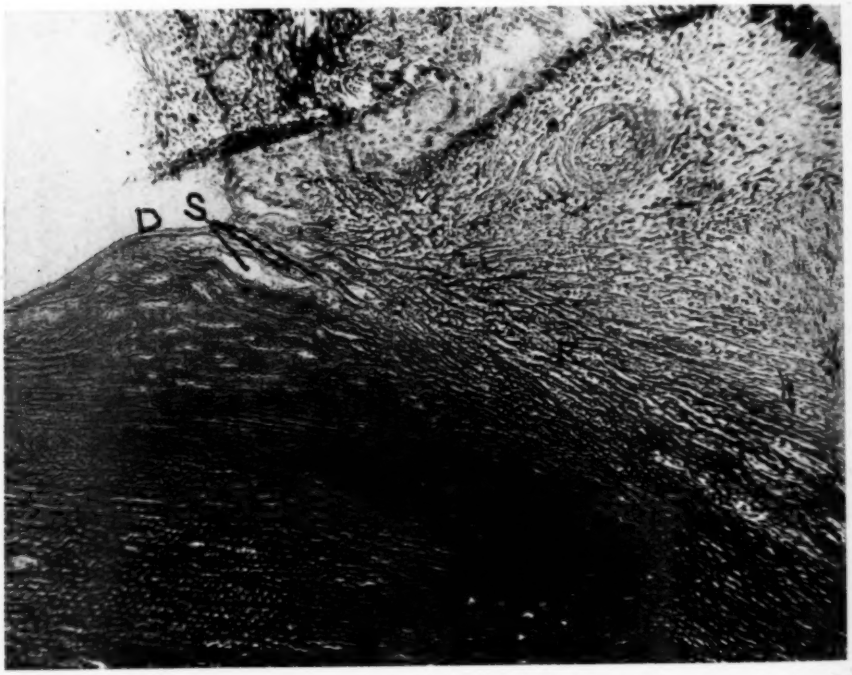

Fig. 2.—Section of hydrophthalmic eye thru base of iris where it is adherent to cornea, showing S, clefts between corneal lamellae, containing F, inflammatory cells, B, branch of anterior ciliary vessels, D, Descemet's membrane. (Van Gieson's stain.)

eye was continually sore and at times became acutely painful, accompanied by fever. The child's general condition was poor; he was pale, had no appetite and had lost weight. The hemorrhage persisted.

On August 18, 1924, the left eye was enucleated and a glass ball implanted in Tenon's capsule. The child's health improved wonderfully, and when seen a month later, he looked the picture of health and had gained several pounds in weight.

CONDITION OF ENUCLEATED EYE.*

The globe was turned over to Dr. H. D. Lamb, whose report follows:

The enucleated eyeball was fixed in Kaiserling's fluid; it was bisected by an incision along the vertical meridian; one half was mounted in gelatin as a museum specimen and the other half including the optic nerve was imbedded in celloidin and sectioned; the sections were stained with hematoxylin-eosin, Van Gieson's and Verhoeff's elastic tissue stains.

Macroscopically, the eyeball (Fig. 1) is seen to be greatly enlarged for a child of 6 years, its measurements being 31 mm. anteroposteriorly by 28 mm. equatorially; the corneal diameter is 19 mm.; a shallow groove or sulcus extends around the ball just posterior to the limbus.

Examination of the bisected globe shows a very deep anterior chamber—about 5 mm. at the center or the deepest part. The anterior chamber contains a small amount of coagulum; the vitreous is quite filled with coagulum and old hemorrhages. The lens, not being affected by the hydrophthalmic changes, appears very small in proportion. The retina is detached completely and has been torn from its usual firm attachment to the optic nerve. The retina has been drawn forwards so as to lie just internal to the ciliary body and posterior to the iris and lens. The papilla shows no cupping.

Microscopically, the anterior epithelium of the lower half of the cornea is two to three times the normal thickness; in this part over about the upper half of the distance between the corneal center and the inferior limbus there is much superficial cystic degen-

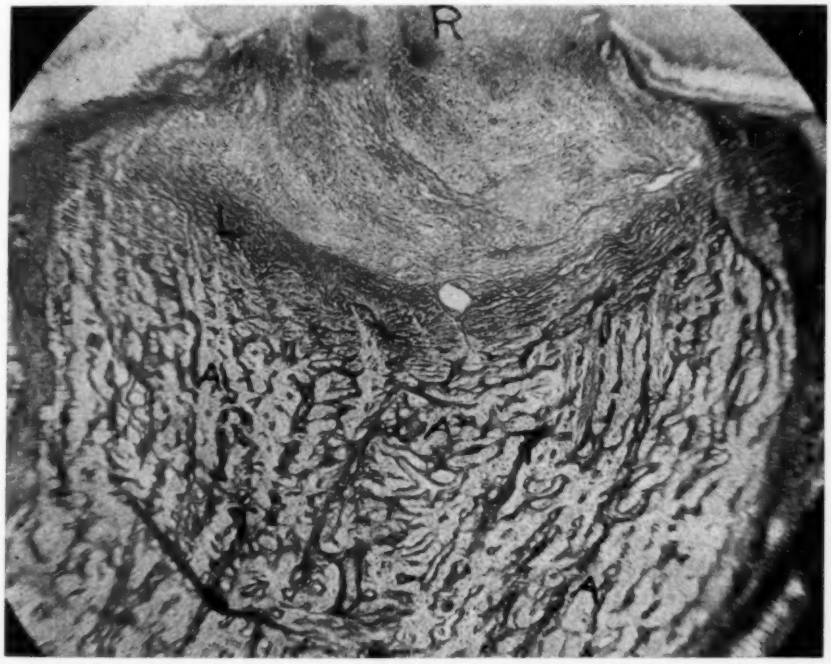

Fig. 3.—Section near middle of papilla of hydrophthalmic eye, showing T, thickened connective tissue septa, A, partially atrophic nerve bundles, L, imperfect lamina cribrosa, R, detached retina pulled forwards. (Van Gieson's Stain).

eration. Most of these cystic spaces have opened externally, giving a shaggy appearance to the cornea at this site. Inferior to this strip, the thickened epithelium contains a few isolated small cystic spaces lying at varying depths below the surface.

Bowman's membrane does not appear in any sections; it has probably been so thinned out by stretching as to disappear; there is never any regeneration of Bowman's membrane.

The substantia propria is everywhere thicker than normal, but most markedly so over the summit of the corneal protrusion and adjoining the limbus. The greatly increased number of young corneal corpuscles testifies to the hyperplasia that has taken place. Diffusely scattered and isolated small lymphocytes and polymorphonuclear leucocytes are seen flattened between the corneal lamellae. Beneath the cystic portion of the anterior epithelium, the hyperplasia of young corneal corpuscles is very great and the connective tissue lamellae are here very narrow, forming scar tissue. The cicatrix is a rather thin crescent shaped strip, which has formed just inferior to the most prominent part of the cornea, insinuating itself just beneath the anterior epithelium. A few capillaries are seen thruout this scar tissue and just beneath it.

These capillaries show a thin loose covering of small lymphocytes with a few polymorphonuclear leucocytes. The elastic tissue stain shows a moderate number of short, delicate, single, thread like fibers, lying between the corneal lamellae and parallel to them. These elastic tissue fibers are scattered irregularly; in some rather large areas (high power) there are none to be made out whereas in other places they are quite numerous. Descemet's membrane and the posterior endothelial layer of the cornea appear normal.

The sclera is, speaking generally, about twice as thick as normal, but in the region of the equator in the lower portion of the eyeball it is three times normal thickness. The inner third of the this thickened sclera is separated from the external portion by a thin linear interspace containing capillaries, chromatophores, small lymphocytes and a few polymorphonuclear leucocytes. This linear interspace is continuous with the choroid posteriorly. Anteriorly it is continuous with the choroid in the upper half of the eyeball, but in the lower portion of the globe where the sclera is thickest it is continuous with the vascular layer of the ciliary body. Thus some of the thickening of the sclera is apparently caused by the internal deposition of new scleral lamellae. In fact these additional lamellae must have been formed within the choroid, for otherwise we would not have the linear interspace derived from the choroid lying between them and the remaining external portion of the sclera. The fibrous lamellae of the sclera show marked hyperplasia of scleral cells.

Fine elastic tissue fibers are diffusely scattered over the outer portion of the sclera. These fibers occupy the periphery of the lamellae for the most part; they are of varying short lengths and run parallel to the bundles of collagenous fibers. In the extrascleral lamellae, lying internal to the choroidal interspace, there are none of these fine elastic fibers. In the outer part of this extrascleral portion there are seen here and there rather long stretches of one to several thick tissue strands, greatly curled and twisted.

The emissary blood vessels and nerves in the anterior and posterior portions of the sclera have a thin, loose covering of small lymphocytes with a few polymorphonuclear leucocytes. Near the superior limbus there are seen bundles of cicatricial fibrous tissue running perpendicular to the direction of the scleral lamellae; this tissue contains a few small blood vessels surrounded by small lymphocytes. This was undoubtedy the site of the trephine opening.

Nothing resembling the filtration tissue of the ligamentum pectinatum or of Schlemm's canal could be made out. There are present (Fig. 2) only good sized clefts between the corneal lamellae containing quite dense number of small lymphocytes and polymorphonuclear leucocytes.

The base of the iris is in contact with the cornea just as in primary glau-

coma. At the anterior end of this adherent portion of iris, there is a sharp bend formed in the anterior surface of the iris by the iris just internal being forced back. The anterior three-fourths of the thickened iris consists of degenerated red corpuscles associated with many chromatophores. On the anterior surface of this portion lies the anterior endothelial layer of the iris. The remaining posterior portion of the iris is very atrophic, and consists of a thin strip of granulation tissue, with large numbers of young fibroblasts and moderate infiltration with small lymphocytes and a few polymorphonuclear leucocytes. On the superior side there is present a long flat fresh posterior synechia. The posterior pigmented epithelial cells have partly disappeared over almost all this adherent portion of the iris. There is present a fairly large cystic space between the posterior epithelial layers of the iris in its inferior portion. The pupil is entirely closed by organizing fibrinous exudate and hemorrhage resting on the anterior lens capsule. In fact, the tissue present in the pupillary area is similar to that of the adherent iris above the pupil, except, of course, that there is no posterior pigmented epithelium in the pupillary area.

The ciliary body shows marked atrophy. Ciliary muscle and stroma are replaced by young cicatricial tissue showing fibroblasts, some chromatophores and a diffuse scant infiltration with small lymphocytes. The ciliary processes are drawn inward into long thin tube like structures.

The folds of completely detached retina lying just inside the ciliary body and behind the lens and iris show marked degeneration. They are separated from the iris and lens in some places by masses of organizing fibrinous exudate; in other places by old hemorrhages containing chromatophores. Behind the lens they are separated from that structure by a thin fresh cyclitic membrane.

The lens shows marked liquefaction in the anterior portion and region of the equator, but only to a small extent posteriorly. The capsule of the lens with the adhering anterior lens epithelium is much wrinkled and drawn away from the lens fibers by the fibrinous exudate, organizing which almost completely surrounds the lens.

The choroid is atrophic, with replacement of normal structure by cicatricial tissue. In some places there is infiltration with small lymphocytes.

The pigmented retinal epithelium is well preserved for the most part; in a few places it shows nodules of varying size made up of pigmented cells derived from this layer. In one place in the lower anterior portion of the fundus there is seen an extensive and thick proliferation of pigmented retinal epithelium. Pigment occurs in very irregular amounts in this latter proliferation, many of the cells containing no pigment whatever.

The optic nerve (Fig. 3) shows no cupping probably because of the imperfect development of the lamina cribrosa. The latter consists of a few strands of connective tissue running entirely across the nerve, altho on one side there are many strands that terminate near the axis of the nerve. Moderate atrophy of the nerve is present with increase in thickness of the connective tissue septa. These changes in the optic nerve are most clearly demonstrated by Van Gieson's stain.

Our study indicates that this was a case of primary hydrophthalmus, which caused general enlargement of the entire globe with deep anterior chamber. Later hemorrhage and inflammation occurred, closing the pupil. This was followed by forward propulsion of the ciliary body and base of the iris by the fluid in the posterior chamber, thereby closing the iris angle. Detachment of the retina was probably caused by hemorrhage from the choroid. The thickening that has occured in the cornea and sclera is probably the result of the diminished tension, present since the operation of sclerocorneal trephining.

Sections in this case were prepared at the Eye Research Bureau, maintained by the Missouri Association for the Blind and the St. Louis University School of Medicine.

Metropolitan Bldg.

MELANOSARCOMA OF THE CHOROID.

FRANK E. BRAWLEY, M. D.

CHICAGO, ILL.

The chief features of such cases are summarized from recent literature. A case is reported in which the eyeball was enucleated and radium used in the orbit. There had been no recurrence after two years. Read before the Chicago Ophthalmological Society, February 16, 1925. (See p. 828.)

When one investigates the literature of this subject, a surprising number of such cases are found. This may be due to the fact that pigment from the choroid is necessarily contained in most intraocular tumors, even in gliomata in children.

The classical evidences of intraocular tumor are hypertension, engorgement of the anterior ciliary veins, retinal detachment, particularly if circumscribed with very limited or no motion of the retina, and a definite shadow with transillumination. S. Hagen, in the Christiania Clinic, in a survey of the subject, concludes that the glaucomatous stage appears usually in one year, or less, after the first symptoms of failing vision, etc., are noted. When metastasis develops, the subject may live from one to eight years. Of twenty-eight cases operated, fifty-eight per cent were cured. Early operation is advisable. Recurrences in the orbit occur only in cases where the tumor has perforated the sclera. The danger of recurrence is greatest in the first six months after operation, and the patient is safe after four years.

In cases where the growths can be seen, they are usually found to be small, circular, fairly sharply defined, and slate gray in color, and have been compared to the color of mercurial ointment. Nettleship, de Schweinitz, Shumway, and R. Foster Moore are agreed that stippling or irregular pigmentation on the surface of the growth indicates activity.

The growth is usually made up of spindle cells with irregular deposits of pigment granules. The average age of incidence is fifty years. Injuries seem to play a role in only ten per cent of the cases.

Ellett[1] reported three cases very similar to the author's case. In each instance an acute glaucoma had developed in an eye previously blind, and each eye was found to contain a sarcoma, altho mention of pigment is made in only one case.

Francis[2] reports a case of malignant melanoma of the choroid, seen first as a circumscribed, yellow-white, raised area, in the macular region. Two years later this region showed a small retinal detachment, with Schiötz 17. After several conflicting opinions and a reduction of tension to 5, an enucleation was done. The pathologic diagnosis was malignant melanoma of the choroid. The most significant features were the gradually decreasing intraocular tension and the absence of the usual evidences of tumor, namely engorged anterior ciliary veins, hypertension, and positive transillumination.

Mr. J. M. H.: Suffered an injury to the left eye 4 years ago. Has been having severe pain in this eye and vision has gradually failed. Three months before I saw him, vision began to fail. Did not consult an oculist, but optician gave him glasses. He was first examined November 7, 1922. There was severe pain, but only slight injection of deep scleral vessels. Finger tension was plus 3. There was no light perception. The lens was completely cataractous, so that no fundus details could be seen. The anterior chamber was practically obliterated. Transillumination of the globe was negative.

Iridotasis was performed, and on November 18, 1922, the tension with Schiötz was 25. There was no pain and the eye was quiet and drainage good. As the tension rose repeatedly and could be controlled only with massage, a possible intraocular tumor was considered and enucleation advised. This operation was performed December 6, 1922. The eyeball was examined by Dr. Edwin F. Hirsch, director of the St. Luke's Hospital Laboratory. A tumor composed chiefly of spindle cells was found near the posterior pole. The tumor was one

cm. in its greatest diameter, and many of the cells were black with pigment.

When healing was complete radium was advised. He received radium in the following dosage, administered by Dr. Wm. L. Brown of the Physician's Radium Association: December 29, 1922, the left orbit was treated with 100 mgs. radium, screened with 0.8 mm. of gold and 2.1 mm. of rubber, for two hours. January 2, 1923, the same treatment was applied. January 3, 1923, 75 mgs. radium was used at one inch distance, for four hours. February 28, 1923, 50 mgs. radium similarly screened was left in the orbit for six hours. March 1, 1923, 50 mgs. radium screened with 0.8 mm. of gold, at 12 mm. distance from the orbital cavity, for three hours.

The right eye showed an irregular hyeropic astigmatism and presbyopia, which were corrected, giving normal vision. The right fundus showed a few fine vitreous opacities but no active uveitis. Search for a focus of infection was negative, except that he was at one time treated for ulcer of the stomach. The orbit shows no recurrence, but the radium has destroyed all the cilia. There was perhaps a larger amount of orbital and lid secretion in this case than is usual, probably due to the radium.

30 N. Michigan Ave.

REFERENCES.

1. Ellett, E. C. American Journal of Ophthalmology, October, 1920, p. 732.
2. Francis, Lee M. American Journal of Ophthalmology, December, 1920, p. 872.

A CRITICAL SURVEY OF NEOPLASMS OF THE CHOROID.

MARY S. KNIGHT, M.D.

ROCHESTER, MINNESOTA.

Reviewing recent work on the origin of melanin it is concluded that the chromatophores of the choroid and skin cannot produce pigment but are only phagocytic cells that ingest the melanin. A case is reported of buphthalmos for which the eye was removed, and studied microscopically. From the study of this specimen and accounts of others it is concluded that the choroid in this and similar cases shows the structure described in neurofibromatosis, the lesion being developmental and not malignant. This work was done at the Mayo Foundation and the report of it submitted as part of the requirements for the Degree of Master of Science in Ophthalmology in the University of Minnesota.

One of the fundamental principles of the science of oncology is that the varieties of tumor to which any type of tissue may give rise will correspond to all the developmental variations which that tissue may assume in the body. Consequently, various types of carcinomas arise from epithelium, and tumors ranging from the fibromas to the varying forms of sarcomas, with many intermediate groups, arise from connective tissue. On this hypothesis a study of the primary tumors of the choroid, usually considered a connective tissue structure, would be expected to include examples of most, if not all, the varieties of neoplasms which might arise in other connective tissue structures of the body. Neither the literature on this subject nor the examination of a considerable number of actual tumors of the choroid has shown this as clearly as would be expected. It is my purpose, therefore, to review this evidence and to report my experience in the examination of the tumors of the choroid which have occurred in the Mayo Clinic, and to furnish some basis for the assertion that perhaps in the final analysis, the melanomas represent the sole type of primary malignant tumors of this structure.

The names that have been given to tumors of the choroid are legion. Fuchs described fourteen varieties of sarcomas and other writers have recommended the inclusion of other types. Parsons seems to refer only to tumors containing melanin or those potentially capable of producing the pigment. He does refer briefly to the possibility of the occurrence of metastatic sarcomas, but is unable to cite an authentic case. In a review of the literature I was unable to find a well

proved case, reported since the publication of his book.

In a previous paper[12] I have discussed the pigmented tumors and indicated that they are probably not sarcomas, but that they are ectodermal in origin. The problem presented by the pigmented tumors of the body has been widely discussed, notably by Knies, Coley, Broders and MacCarty, and Knight. The earlier writers, when describing the pigmented tumors of the choroid, sometimes called them

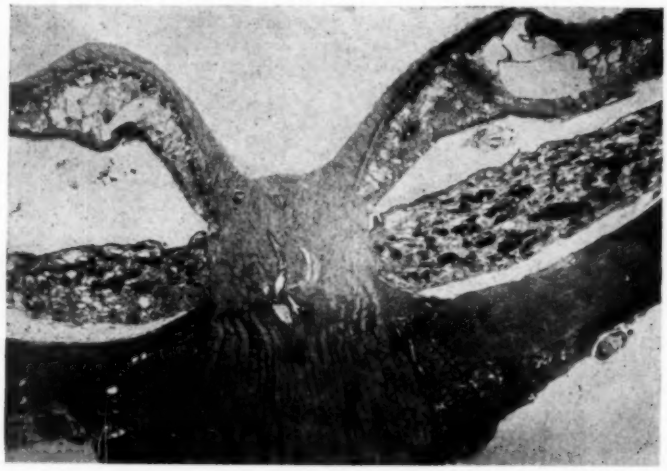

Fig. 1.—Hemangioma of choroid encircling the papilla (x 15).

carcinomas or epitheliomas, and they frequently referred to the epithelial character of the cells.[13, 31] Later when the same structural characteristics were described, the author's uncertainty was hidden under the term "endothelioma."[1, 10, 21] Recently, as tho by mutual consent, all of this group of pigmented and nonpigmented tumors of the choroid, have been called sarcoma.

Wherever studied, it was found that melanin was produced by ectodermal cells. Smith, working at Johns Hopkins University, studied the production of melanin granules in the pigment epithelium of the eye of the chick embryo. He used mesoblastic choroidal cells as controls, and no pigment was ever produced in them. However, they did phagocytize pigment granules which were freed by the breaking of epithelial cells in preparing the specimens. Results subject to a similar interpretation have been noted in investigations on the skin with "dopa," a substance allied to tyrosin, which was isolated from the broad bean by Bruno Black, Professor of Dermatology at Zurich. This substance in weak solution, when applied to bits of fresh skin, turned them grey, and microscopically the change was seen to be due to the appearance of melanin granules in the cells. The reaction occurs in cells capable of producing melanin, such as those of the epidermis in the fetus, even before pigment has appeared, as well as in those of the skin of adults. The color of the cells of the pigmented nevus and the pigment epithelium of the retina is increased when they are treated with dopa, but the chromatophores of the dermis and of the choroid do not give the reaction. They are in all probability incapable of producing pigment, that is, are not true melanoblasts, but only phagocytic cells which ingest the melanin. The malignant, pigmented new growths, occurring in the skin, can often be seen to rise in the epidermis and are definitely epithelial in origin. The tumors arising in the choroid that have similar histologic characteristics, cannot reasonably be assigned another name.

A case reported by Parsons is of interest here, especially if another interpretation is given than that used by him. The left globe of a man, aged

forty-five, was removed on account of proptosis and enlargement. Enucleation of this eye had been advised two years before, because of loss of vision and the presence of inflammation. The globe was filled with a deeply pigmented growth which perforated the sclera and projected backward around the optic nerve. The patient died one year later, and at necropsy a soft white tumor was found in the right chest, invading the third, fourth, and fifth ribs, the pleura, and lungs, mediastinal nodes and thymus. There was also a few bile stained, nonpigmented nodules in the liver. The intraocular growth was practically all necrotic. The extraocular extension was made up of poorly staining large polygonal cells with large nuclei and bundles of spindle-shaped cells, which contained very little pigment. The growth in the chest was diagnosed squamous cell carcinoma and the nodules in the liver spindle cell sarcoma. Parsons believed he was dealing with two independent tumors, a necrotic melanosarcoma of the choroid with metastasis in the liver and a squamous cell carcinoma in the chest, the primary focus of which he did not locate. If the melanotic neoplasms are ectodermal in origin, they might be capable of producing squamous cells, and melanin has been found in squamous cell epitheliomas. Metastatic foci are frequently more rapidly growing than the primary growth, but they occasionally grow more slowly and become more highly differentiated than the original tumor. It is possible that the nodules in the liver and chest were metastatic growths from the tumor in the eye.

Aside from these sarcomas, or malignant melanomas, as I would prefer to call them, and metastatic carcinomas, Parsons mentions very briefly dermoids, myomas, hemangiomas and plexiform neuromas. He had never seen a dermoid, altho he knew of no argument against their occurrence in the choroid, and the literature afforded him only one example, which occurred between the retina and choroid. He says that myomas, also rare, occur in front of the equator of globe, and, arising from backward extensions of the ciliary muscle fibers, really belong to the ciliary body.

In about 200 eyes examined microscopically during the last three years I found an example of each of the two remaining types. Both are rare tumors and ordinarily would not be met with in such a short series. The hemangioma involved the choroid around the posterior pole and encircled the optic nerve (Fig. 1). It was not encapsulated, but was fairly well limited both macroscopically and microscopically. Temporally it extended 5 mm.

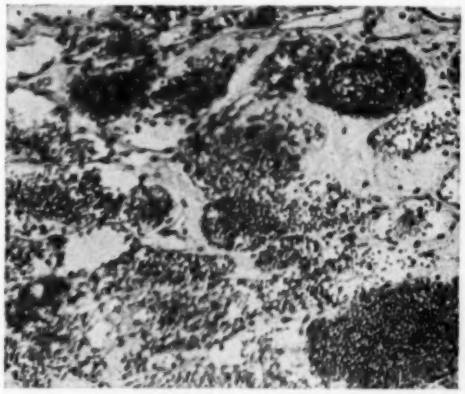

Fig. 2.—Hemangioma of the choroid (x 150).

from the optic disc, and nasally less than that. Grossly the lesion resembled a massive subretinal hemorrhage. Microscopically it was composed of large, thin walled, endothelial lined spaces filled with well preserved erythrocytes (Fig. 2). There were no evidences of thrombosis. There was scanty connective tissue in the walls of the spaces and practically no pigment within the growth. The hemangioma extended to the suprachoroidea, and in most places the inner surface was covered by the choriocapillaris. Occasionally it extended to the lamina vitrea, but this and the pigment epithelium were intact. The growth did not infiltrate sclera, retina, or papilla.

Most of the cases of hemangioma of the choroid reported in the literature were associated with congenital "port wine" nevi of the lids and same side of the face. The histologic picture was the same as described in this case except that some contained lamellae of

bone, between the blood spaces or over the inner surface of the tumor. In all the cases reviewed, the growth was limited to the choroid and was strictly a developmental anomaly rather than a true neoplasm.

Parsons knew of one case of plexiform neuroma of the choroid, and that had been reported by Collins. It occurred in a girl aged fourteen. The right eye was enlarged at birth. At examination the right upper lid and skin covering the orbit were hypertrophied and there was a soft, doughy swelling over the temporal fossa. As the buphthalmic eye was irritable and the vision reduced to almost nothing, the eye and a wedge shaped portion of the upper lid were removed. The tumor in the lid was identified microscopically as a neurofibroma, and the thickened choroid showed a similar structure. An illustration which accompanied the case report resembled the microscopic picture of the choroid in a buphthalmic eye I examined.

Buphthalmos, occurring coincidently with neurofibromatosis of the orbit or eyelids, had been reported previously in the literature. Sachsalber, who was the first to recognize the interrelation of the two conditions, published in 1898 a detailed account of a case, and a discussion of the etiology. His patient was seven years of age. The left side of the face was enlarged and deformed at birth, and this condition progressed.

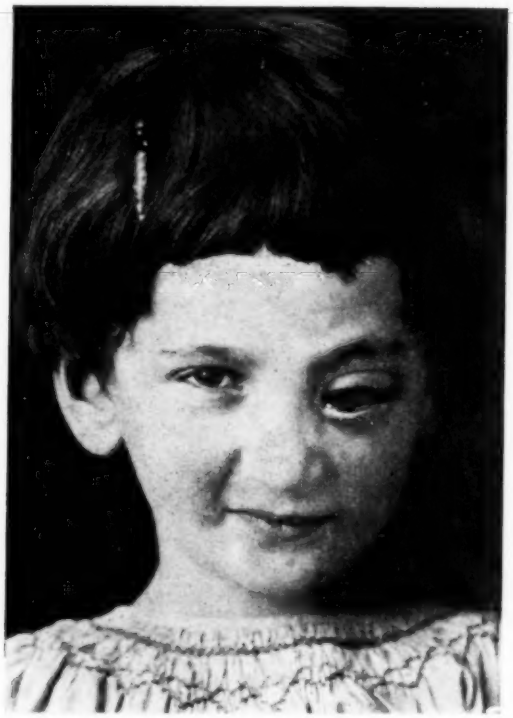

Fig. 3.—Five year old girl with enlarged left eye.

The deformity of the face was caused by malformation of the bones and a tumor. The left eye was enlarged. The globe and part of the tumor were removed and examined microscopically.

In 1903, Snell and Collins reported three cases of plexiform neuroma in the orbital region, but in only one was the eye buphthalmic. The next year Rockliffe and Parsons published the case record of a girl six years of age, whose right eye was closely associated with a large pulsating tumor of the orbital region, which was found to be a plexiform neuroma. In this case, however, the eye was not enlarged. Sutherland and Mayou, in 1907, reported buphthalmos, associated with enlarge-

ment of the right side of the face and thickening of the branches of the third division of the fifth nerve. No pathologic study was made. In Weinstein's case, that of a boy aged sixteen, a piece of the hypertrophied left upper lid was excised, but the buphthalmic eye was not enucleated. The tumor of the lid presented the typical microscopic appearance of neurofibroma. The same year Cabannes reported a case of hemihypertrophy of the face associated with buphthalmos, occurring in a girl, six years of age. The eye was enucleated and studied microscopically; no pathologic study of the tumor involvement of the face was made, and the correlation of the two conditions was only suggested by the author.

In 1913, Murakami carefully reviewed the literature, and reported in detail the pathologic findings in a buphthalmic eye removed by Professor Sato. Numerous other ocular lesions in connection with neurofibroma of the lid or orbit and with generalized neurofibromatosis have been reported, but this study is not concerned with them. In all, six eyes were examined microscopically, but only in the case reported by Batten and Collins was the lesion of the choroid similar in structure to the orbital tumors. A case reported by McKay in 1875 as one of "general sarcoma of the choroid, probably congenital" most likely belongs to this group.

In the literature there was no instance of this type of lesion involving the choroid without an associated orbital tumor. In my case, the lesion was confined to the choroid, and even at operation no orbital tumor was found.

REPORT OF A CASE.

A five year old girl, of Jewish parentage, was first examined May 20, 1924, because of an enlarged left eye (Fig. 3). This eye was larger than its fellow at birth and had gradually increased in size. The vision was 6/10+ in the right eye; the left eye was blind. The right orbit and eyeball were normal, the left orbital aperture was larger and lower than the right, and the left globe was very much enlarged. The left lids were greatly stretched and somewhat edematous. The right cornea measured 11 mm. in diameter; the left measured 16 mm. in the horizontal meridian. Exophthalmometer readings were 15 mm. in the right eye and 20 mm. in the left eye. The left eye diverged fully 30 degrees, and its increased size restricted its motility. The cornea was diffusely hazy and in the center there was a denser opacity measuring 3 by 5 mm. The iris was greenish grey, and tremulous. The lens was partially dislocated, suspended only by the zonular fibers above and nasally. It was small and greenish yellow. The tension of the left eye was increased to palpation. The child was well nourished and in good health. Delivery had been normal and at full term. The serum Wasserman reaction was negative. Neurofibromatosis was thought of at the time of examination, but no tumors or developmental anomalies other than the enlarged eyeball and stretched lids, were noted in the routine inspection.

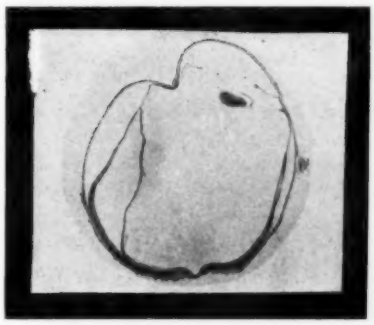

Fig. 4.—Stained section.

The left eye was enucleated because it was blind, unsightly, and steadily increasing in size. Nothing unusual was encountered in the orbit. A glass ball was implanted in Tenon's capsule. There was slight chemosis and edema of the lids for several days after operation, but not more than is generally produced by glass ball implantation. The conjunctival wound promptly healed. The edema subsided within a week.

On gross examination the globe was enlarged, especially in the anteroposterior diameter, which measured 35 mm.,

and was practically barrel shaped. The average transverse diameter was 27 mm. The cornea was also enlarged, measuring 15 by 16 mm. It was hazy, but not uniformly so, for in the center the haziness amounted to actual opacity. The anterior chamber was deep; the pupil large, oval and eccentric. The lens was small, mobile and did not fill the entire pupillary area. The posterior part of the globe was smoothly rounded.

The globe was sectioned along the upper and lower margins of the cornea.

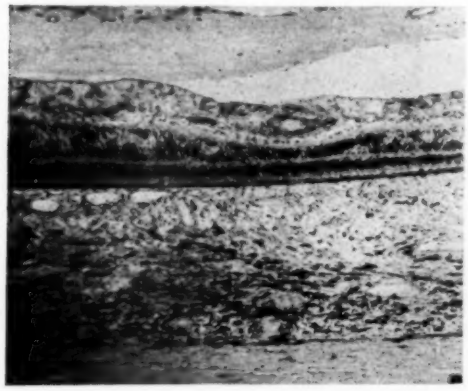

Fig. 5.—Thickened choroid near the posterior pole (x 50).

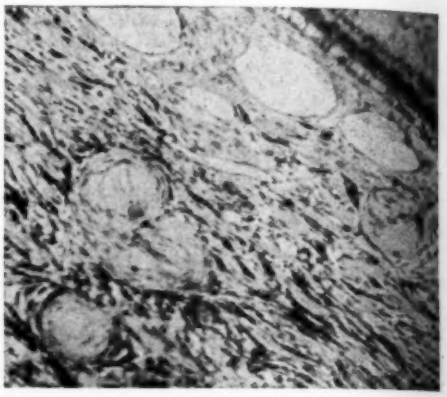

Fig. 6.—Showing ovoid bodies in choroid (x 150).

The vitreous was fluid and cloudy. The retina was opaque, yellowish and traversed by bands and folds. The optic disc was large and excavated, but the excavation appeared to be funnel shaped and the edges of the nerve-head were not undermined. Anterior to the equator the choroid appeared thin, atrophic and, in some places, almost depigmented. Behind the equator the choroid was dark, fleshly, and several millimeters in thickness. The lens was small and eccentric, and only a few long fibers anchored it on the temporal side. The central portion of the specimen was dehydrated in alcohol, embedded in celloidin, hardened in chloroform vapor, cleared in cedar oil, cut dry on a paraffin microtome, the oil extracted in 80 per cent alcohol, and the sections, which were 15 microns thick, stained by several methods. Hematoxylin and eosin staining was used for the routine examination (Fig. 4).

The cornea, which was increased in diameter showed reduced but uniform thickness. The epithelium gave the impression of slight irregularity, but this may have been due to the absence or very imperfect formation of Bowman's membrane. In some areas there was a lighter stained, irregular band, beneath the epithelium, but nowhere was there an even, definitely formed membrane. The substantia propria was fairly normal. The lamellae were evenly arranged in parallel layers and showed no infiltration except near the limbus, where strands of vessels invaded all except the deepest layers. Descemet's membrane and the endothelium were present except near the angles of the chamber, where the root of the iris was adherent to the cornea. There both layers were reflected over the anterior surface of the iris and extended medially half way to the pupillary margin. Descemet's membrane frayed out shortly before the endothelium disappeared.

The anterior chamber was about the usual depth, but if the iris had been in its normal place the chamber would have been very deep. The angles of the chamber were blocked by the peripheral anterior synechia.

The sclera was thinner even than that of young eyes, but the thinning was uniform. The connective tissue bundles were normal in appearance and arrangement. The nerves and blood vessels showed no anomaly.

The iris was thin and fibrous. The peripheral portion of the anterior surface, beyond the adhesions, was covered by the continuation of Descemet's membrane and endothelium, and the medial

part by uveal pigment, which had been dragged around the pupillary margin. In sections taken on one side of the pupillary opening, the ectropion of the uvea formed a long line of pigment in the center of the anterior surface of the iris. There were very few vessels in the iris and these were thin walled and slit like, instead of having normal well developed walls. The circular muscle was well developed, but the pupillary end was rolled forward along with the ectropic uvea. The posterior pigment epithelium formed a thin, flat, brown band, which did not show the usual serration. There was scarcely any stromal pigment present.

The lens was small, the transverse diameter being about two-thirds that of the pupil, or one-third the diameter of the ciliary ring. The lens capsule was intact. There was a layer of epithelial cells under the posterior capsule, but they were few in number and inconspicuous when compared with the cells of the anterior epithelium. The cortical area was filled with a substance largely homogeneous, which took practically no stain, but in places was marked off into fibers by fine pink staining lines. Many of these fibers, especially in the equatorial region and some beneath the anterior capsule, contained faintly staining nuclei. The epithelial nuclei beneath the capsule stained deeply. The medulla, which stained deeply and contrasted sharply with the cortex, was also homogeneous. The line of demarcation between the two was irregular and broken. Along this line were several flat, granular, deeply staining calcium deposits. On the temporal side of the lens the zonular fibers could be traced from the capsule to the ciliary processes, but on the nasal side the fibers were curled up against the capsule. The ciliary processes on the nasal side of the globe were small and few in number as compared with the opposite side, and the ciliary body also was much flatter there.

The ciliary body was thinner than normal on both sides; and did not taper posteriorly, but was continuous with a thickened choroid. The processes were small and few in number. Blood vessels were not numerous. In contrast to the iris, the stroma was rather heavily pigmented. The radiating portion or Brücke's muscle was well defined, but Müller's fibers were indistinct, and practically absent on the side where the lens was not attached. The ciliary epithelium was normal in appearance.

The thickened choroid increased in width toward the posterior pole, eventually reaching six or eight times the width of the normal tissue (Fig. 5). The great thickening was due to an excess of connective tissue; the whole layer was pigmented, tho not heavily, but the total amount of pigment was much larger than that usually found in the choroid. The vessels were easily seen, for they stood open, but they were greatly decreased in number. By far the largest number were situated in the inner half of the layer near the retina. Most of the lumina

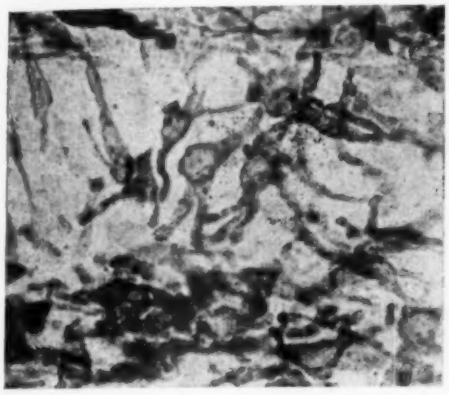

Fig. 7.—Fixed frozen section of the choroid showing branched pigment cells. Stained with Scharlack R and hematoxylin (x 400).

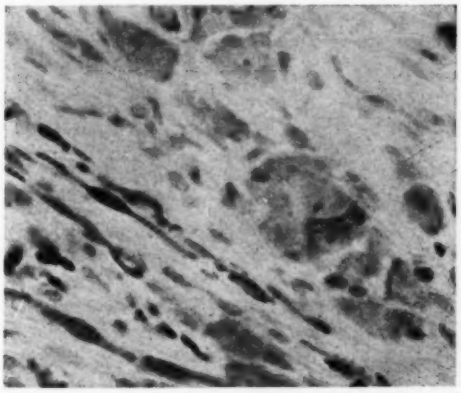

Fig. 8.—Groups of ganglion cells in choroid stained with thionin (x 500).

were large, the walls were thin, and only a very few arteries were found near the suprachoroidea where the large vessels should be. Instead of a vascular layer, the choroid was a solid, pigmented tissue. In it were numerous small, round or oval, laminated bodies, which were best developed in the outer half of the layer. They seemed to be made up of concentric layers of fibers or cells (Fig. 6). As a rule their centers were acellular, but a few flattened nuclei lay between the outer lamina and occasionally a little brown pigment was also caught between them. The pigment was banked

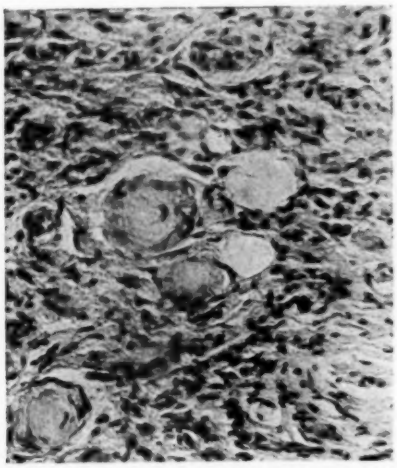

Fig. 9.—Specimen from Dr. Brownlow Biddell's case (x 150).

around the edges of most of these bodies. Some of them seemed to have a fibrous stalk. In frozen sections the branching character of the pigment cells was well seen (Figs. 7 and 8). Thruout the choroid, especially in the equatorial region, were large cells resembling ganglion cells. Sometimes they occurred singly, sometimes in small groups, and in certain fields were very numerous (Fig. 9). They had abundant cytoplasm and large round nuclei in which a single large nucleolus was seen. These cells stained well with thionin, and a few seemed to show Nissl granulation.

The lamina vitrea was very thin and could not be followed, but seemed to be present only around the posterior pole. The pigment epithelium, however, could be traced thruout and appeared fairly normal. The retina itself was very poorly formed. On both sides of the disc the various layers of the retina could be distinguished, but these faded out toward the equator, and anterior to it the characteristic arrangement of the retina was not seen. Near the disc the layer of rods and cones and the external limiting membrane were well defined. The nuclear layers were wide and frayed, and the internuclear layer poorly defined. The ganglion cells and fiber layer were replaced here by connective tissue. Elsewhere the retina was made up of a broad network of fine fibers and small nuclei which were most numerous near the pigment epithelium. In some places the arrangement of the nuclei suggested a division into two layers. Lying along the surface of the retina were strands which evidently represented the vitreous. A large one was made up of faintly staining homogeneous material in which were thin walled vessels and scattered strands of cells. The optic disc contained a deep, funnel shaped cup, the apex of which seemed to perforate the lamina cribrosa; elsewhere its fibers were not greatly displaced. The optic nerve had been cut very close to the globe, but in the scleral portion no trace of nerve fibers was seen.

DISCUSSION.

There was no sign of inflammatory exudation in the eye and none of the nerves in the sections appeared to be abnormal. The ciliary nerves were greatly thickened in the eyes described by Sachsalber, Murakami, and Snell and Collins. Collins found hypertrophic nerve fibers in the cornea, represented by tracks of elongated cells, in both of the eyes he examined. There were a few tracks of cells in the periphery of the cornea in this case, but lumina filled with erythrocytes were so numerous along them, that there could be little doubt that they represented only vessel walls.

The changes in the choroid in these buphthalmic eyes are variously described. Sachsalber found the choroid thickened because of an increase in connective tissue and pigment bearing cells, the vessels reduced in number and the choriocapillaris largely destroyed, all of which findings agree with mine. However, he does not mention the ovoid bodies found by Collins. Murakami found practically the same changes in a localized area of

thickening on the temporal side of the posterior lobe. The description of the choroid in the case reported by Batten and Collins corresponds to that given for the case here reported, except that the numerous ganglion cells were not noted by Collins. He describes the ovoid bodies as nerve end organs and calls them Pacinian corpuscles.

cular mantles derived from the mesoderm surroundings the neural tube and its outgrowths. In 1919, Mallory showed that the socalled "dural endotheliomas," which arise from the arachnoid, are really tumors of the connective tissue series, whose type cell is the fibroblast. These he called arachnoid fibroblastomas to distinguish them from other connec-

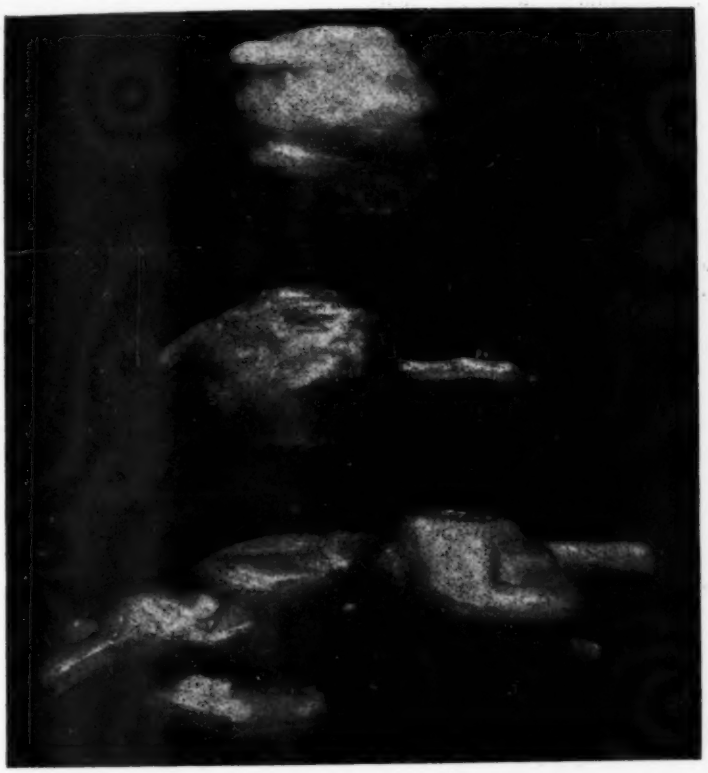

Fig. 10.—Wax reconstruction of the laminated ovoid bodies in the choroid (x250).

The choroid contains a rich plexus of both medullated and nonmedullated nerve fibers which is derived from branches of the long and short ciliary nerves. Ganglion cells are found in groups at the nodal points of this plexus, and sometimes singly along the vessels. These fibers supply the choroidal vessels to their finest ramifications and are regarded as vasomotor in function. The choroid contains no sensory fibers; at least inflammatory processes in this structure are painless so that the presence of sensory nerve end organs is difficult to explain.

The choroid is analogous to the pia arachnoid of the brain, both being vas-

tive tissue tumors. He showed that the tumors arising from the perineurium of the peripheral nerves which have been called fibroma neuroma, fibroma molluscum, plexiform neuroma, and, when multiple, von Recklinghausen's disease, are neoplasms of the same group; and closely allied to those arising from the arachnoid, which is a structure comparable to the perineurium.

The cells of the arachnoid tumors tend to form whorls, wrapping themselves around each other or around collagen strands. These whorls undergo degenerative changes, and in some tumors become calcified to form the psammoma bodies. If the process has

not gone on to calcification the whorls appear as hyalin, laminated bodies in which some of the cells may still stain. To my mind, the ovoid bodies found in the choroid are structures similar to the psammoma bodies, or their hyalin anlage, in the socalled dural endotheliomas.

Serial sections of the eye were stained with hematoxylin and eosin, and wax model reconstructions made of these bodies (Fig. 10). They were roughly ovoid in shape, one main diameter being about half as great as the other two. They varied somewhat in size, the long diameter of those reconstructed ranging from 90 to 135 mm. A short fibrous stalk was frequently seen in one section, but could never be traced into the adjoining sections nor made to connect with anything that looked like a nerve. Often two or three of these bodies lay in contact. They stained poorly with eosin, pale salmon pink with van Gieson's stain, and did not take Bielschowsky's stain. If they contained nerve fibers these should have been stained by the silver. Pacinian corpuscles are microscopically visible, hence much larger structures than those seen in the choroid. Lewis and Stöhr describe them as 0.5 to 4.5 mm. long and from 1 to 2 mm. wide, while the average length of the choroidal bodies was 0.1 mm. In the interior of the tumor removed from the orbit in the case reported by Rockliffe and Parsons, the latter found a few curious bulbs which he presumed were nerve end organs of some kind. The picture of them accompanying the text shows the same structure as the photomicrograph of the choroidal bodies shown here (Fig. 6). Similar structures were also found in a section of an orbital tumor which came to my notice thru the kindness of Dr. Riddell of the Glasgow Eye Infirmary (Fig. 9). This tumor was removed from a man, forty-five years old, who was born with an enlargement of the left lower lid. At the age of twenty the left eye was eviscerated in Berlin. The left lids, which were enlarged and dependent, were a source of embarrassment to him. In the summer of 1924, Hutton, in Western Infirmary, Glasgow, removed both lids and stitched the skin margins together over the orbit. In the piece of tissue examined, the tumor, which was fibrous and fairly cellular, had invaded the fat. The cells had a tendency to form in whorls, which became hyalinized, most of the nuclei disappearing. These hyalin whorls were not as uniform and well formed as the choroidal bodies, but on the average were of about the same size and shape. This was confirmed by following individual bodies thru serial sections of the tumor.

Parsons and Collins were justified in calling this condition of the choroid plexiform neuroma, if by that term a tumor of connective tissue origin is meant, but I cannot agree with the interpretation of the ovoid bodies as nerve end organs. They impressed me as being only hyalinized whorls of connective tissue cells.

SUMMARY.

The occurrence of buphthalmos with neurofibromatosis around the orbit has been noted and recorded several times in the literature. Both conditions were usually congenital. In one case the choroid was thickened because of an increase in connective tissue, and contained hyalin ovoid bodies similar to those found by another author in the orbital tumor which was diagnosed as plexiform neuroma. These ovoid bodies were called Pacinian corpuscles, but almost surely are hyalinized whorls of fibroblasts rather than nerve end organs.

The eye studied here was enlarged at birth, and clinically was a typical example of buphthalmos. Altho it was not associated with neurofibromatosis about the orbit or elsewhere, so far as is known, microscopically the choroid showed the same structure as had been described for that disease. The rest of the eye was not involved in the process but showed evidences of arrested development: vessels and fixed tissue cells in the remnants of vitreous, incomplete differentiation of the retina, small lens, and absence of Bowman's membrane. This lesion in the choroid could also be grouped with the developmental anomalies, for it was certainly congenital, and showed none of the characterictics of malignancy. It was limited to the choroid; the cells were well differentiated and showed no evidences of active proliferation.

Mayo Clinic.

BIBLIOGRAPHY.

1. Alling, A. N., and Knapp, A. A Case of Ring Sarcoma of the Ciliary Body. Tr. Am. Ophth. Soc., 1909-1911, v. XII, pp. 778-786.
2. Broders, A. C., and MacCarty, W. C. Melanoepithelioma: A Report of Seventy Cases. Surg., Gynec. and Obst., 1916, v. XXIII, pp. 28-32.
3. Cabannes, C. La buphtalmie congénitale dans ses rapports avec l'hémihypertrophie de la face. Arch. d'Ophtalmol., 1909, v. XXIX, pp. 368-380.
4. Coley, W. B., and Hoguet, J. P. Melanotic Cancer: With a Report of Ninety Cases. Tr. Am. Surg. Assn., 1916, v. XXXIV, pp. 319-383.
5. Collins, E. T., and Batten, R. D. Neurofibroma of the Eyeball and Its Appendages. Tr. Ophth. Soc. U. Kingdom, 1905, v. XXV, pp. 248-257.
6. Collins, T., and Paton, L. Angioma of the Choroid. Am. Jour. Ophth., 1919, v. II, p. 437.
7. Fehr, O. Ein Beitrag zu den Augen-Veränderungen bei der sog. multiplen Neurofibromatose. Centralbl. f. prak. Augenh., 1913, v. XXXVII, pp. 233-243.
8. Fuchs, E. Exhibition of Specimens. Smallest Sarcoma of the Choroid. Tr. Am. Ophth. Soc., 1909, v. XII, pp. 786-790.
9. Henderson, E. E. Angioma of the Choroid. Brit. Jour. Ophth., 1920, v. IV, pp. 373-374.
10. Knapp, A. Report of a Case of Flat Sarcoma (Endothelioma) of the Choroid—Death One and One-half Years Later from General Metastases. Arch. Ophth., 1905, v. XXXIV, pp. 62-68.
11. Knies, M. Sixteen Cases of Sarcoma of the Choroid, With Epicritic Remarks. Arch. Ophth., 1877, v. VI, pp. 376-431.
12. Knight, Mary S. Melanotic Neoplasms of the Eye. Jour. Am. Med. Assn., 1924, v. LXXXIII, pp. 1062-1068.
13. Laurence, J. Z. Tumor of the Choroid. Tr. Path. Soc., London, 1867, v. XVIII, p. 223.
14. Lewis, F. T., and Stöhr, P. Textbook of Histology. Philadelphia, P. Blakiston's Son and Company, 2nd ed., 1913, p. 161.
15. Love, J. M. Simple Angioma of the Choroid. Arch. Ophth., 1914, v. XLIII, pp. 607-617.
16. McKay, R. J., and Eno, H. C. A Case of General Sarcoma of the Choroid, Probably Congenital. Tr. Am. Ophth. Soc., 1875, pp. 345-348. (Eleventh annual meeting.)
17. Mallory, F. B. The Type Cell of the Socalled Dural Endothelioma. Jour. Med. Res., 1919-1920, v. XLI, pp. 349-364.
18. Murakami, S. Zur pathologischen Anatomie und Pathogenese des Buphthalmus bei Neurofibromatosis. Klin. Monatsbl. f. Augenh., 1913, v. XVI, pp. 514-520.
19. Norris, W. F., and Oliver, C. A. System of Diseases of the Eye. Philadelphia, J. B. Lippincott Company, 1897-1898, v. I, p. 261.
20. Parsons, J. H. The Pathology of the Eye. G. P. Putnam's Sons, New York, 1905, v. II, pp. 493-533.
21. Parsons, J. H. Some Anomalous Sarcomata of the Choroid. Tr. Ophth. Soc. U. Kingdom, 1905, v. XXV, pp. 193-237.
22. Quackenboss, A. Angioma of the Choroid. Tr. Am. Ophth. Soc., 1906-1908, v. XI, pp. 557-562.
23. Rockliffe, W. C., and Parsons, J. H. 4. Plexiform Neuroma of the Orbit. Tr. Path. Soc., London, 1904, v. LV, pp. 27-39.
24. Sachsalber, A. Ueber das Rankenneurom der Orbita mit sekundärem Buphthalmos. Beitr. z. Augenh., 1897, v. XXVII, pp. 1-43.
25. Salzmann, M. The Anatomy and Histology of the Human Eyeball in the Normal State; Its Development and Senescence. E. V. L. Brown, Chicago, 1912, p. 56.
26. Smith, D. T. The Pigmented Epithelium of the Embryo Chick's Eye, Studied in Vivo and in Vitro With Special Reference to Origin and Development of Melanin Pigment. Bull. Johns Hopkins Hosp., 1920, v. XXXI, pp. 239-246.
27. Snell, S., and Collins, E. T. Plexiform Neuroma (Elephantiasis Neuromatosis) of Temporal Region, Orbit, Eyelid, and Eyeball. Notes of Three Cases. Tr. Ophth. Soc., U. Kingdom, 1903, v. XXIII, pp. 157-177.
28. Spencer, W. G. The Bradshaw Lecture on Melanosis (Melanin: Melanoma: Melanotic Cancer). Brit. Med. Jour., 1923, v. II, pp. 907-913.
29. Sutherland, G. A., and Mayou, M. S. 2. Neurofibromatosis of the Fifth Nerve With Buphthalmus. Tr. Ophth. Soc., U. Kingdom, 1907, v. XXVII, pp. 179-181.
30. Van Schevensteen, A. Les Complications Oculaires dans la Neurofibromatose Généralisée. Ann. d'Ocul., 1918, v. CLV, pp. 328-335.
31. Weeks, J. E. A Case of Epithelioma of the Choroid. Opth. Rec., 1907, v. XVI, pp. 163-165.
32. Weinstein, A. Ein Fall von Buphthalmus mit kongenitaler Hypertrophie des Oberlides. Klin. Monatsbl. f. Augenh., 1909, v. XLVII, pp. 577-581.

CONICAL CORNEA.

CHICAGO, ILL.

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The historical development of our knowledge of conical cornea is traced since it was first observed nearly two centuries ago. The various methods of treating it are noticed with approval of the conservative views of Beard. An illustrative case is reported. Entrance Thesis, Chicago Ophthalmological Society.

Corneal ectasias have long excited the interest of the ophthalmologist. The diagnosis being relatively simple, especially in advanced cases, the pathology and treatment have engaged the attention of most observers. Conical cornea falls within this class of ocular pathology. Demours[1] observed the condition in 1747, and it was described by Scarpa[2]. For years there was a question as to whether the cone was solid and thickened, or simply a bulging forward of thinned corneal tissue. Sir William Adams[3] described the condition as a morbid thickening and growth of the substance of the cornea, while Himly gave it the name "hyperkeratosis." Jäger[4], in 1830, on dissecting such an eye, found the cone very thin but the periphery of the cornea thickened. Salzmann's[5] histologic examination of a case showed a thinning of Bowman's membrane with many gaps in it, and a gap in Descemet's membrane near the center of the ectasia. The deeper layers of the cornea, near the center of the conus, tended to split. The region of the ectasia was half the thickness of the normal cornea. Other changes in the eyeball were described as anomalies of development.

The disease usually manifests itself between the fifteenth and thirtieth year, growing gradually more pronounced, until a point is reached at which it becomes stationary, with the formation of a scar and diminution of[6] vision, but without spontaneous rupture of the weakened tissue or increase in intraocular tension. However, cases do occur in which the symptoms appear later in life and others in which no scar is ever noted while under observation. The various conditions with which it has been associated are very often absent—injury, anemia, or disease of Descemet's membrane.[6] It seems therefore, more logical to assume a developmental defect. Bowman's four cases in one family strengthens this view of developmental defect, while it is the belief of Sattler[7] that keratoconus is a congenital anomaly, like high myopia and keratoglobus, without intraocular changes. The cornea probably reaches the limit of its growth in the first twelve months[5], but production of the fibrous elements continues. If for any reason this further development of fibers near or at the center does not occur, that area might be unduly weakened and become ectatic[8]. It has also been pointed out that this area is the farthest removed from the blood supply.

Comparatively little is as yet known regarding the physiologic and pathologic changes which take place during adolescence, despite the accumulating knowledge of such processes and the action of the various agents secreted by the hormone producing glands at that time. If conical cornea is a congenital anomaly, or the result of birth injury or of a low grade descemetitis, the appearance of the distressing symptoms at or shortly after puberty might very well be explained on a basis of some metabolic or nutritional change at that period of life. No extensive work has as yet been done on this phase, altho it is very suggestive as pointed out recently by Imre[9].

The diagnosis of beginning conical cornea may be very difficult, and unquestionably many very early cases are overlooked, for the cornea is perfectly transparent. Examination of the media with the ophthalmoscope reveals, against the red background of the fundus, shadows which shift rapidly. With Placido's disc,[10] distorted rings are mirrored on the cornea. By observing the corneal reflexes, especially the behavior of the posterior, a sudden change in its appearance will be noted at the point of greatest concavity, which corresponds to the cone. With the aid of focal illumintion, fine gray lines are noted at the apex of the cone. Occasionally a yellowish or greenish ring is found surrounding the apex of the cone. The

true nature of these Fleischer rings is not known, being either a deposition of hemosiderin or of pigment.[11] Rarely a pulsation in the apex of the cone can be demonstrated[12]. Not until well advanced does the cornea assume the shape of a cone, but Pickford[13], in 1843, called attention to the glistening appearance of the cornea, which he described as a "dew drop or piece of solid crystal embedded in the cornea," which he noted in the early cases.

The treatment of this disease has been directed toward the correction of the visual symptoms surgically and mechanically, without attempting much locally or generally. Mackenzie's dictum was that he had never known conical cornea lessened by any remedy, internal or external[14]. Drugs used locally have never given satisfactory results. The true nature of the condition not being known, internal medication has been limited to tonics except where some general condition of the patient, such as anemia, has warranted more specific treatment, and in those cases where glandular extracts have been tried, without materially changing the course.

Since 1817, when Sir William Adams[15] advocated "breaking up the crystalline lens in order that the rays of light might fall upon the retina," particularly in cases which must have been myopic, many procedures have been advocated to correct the deformity of the cornea or alleviate the visual symptoms. In 1840, Tyrrell[16] advised removing the pupil from beneath the scar by allowing the iris to prolapse thru an incision at the limbus. Critchett[17] modified this procedure by ligating the herniated iris. Bowman[18] attempted to form the pupil into a stenopaic slit. Dix[19], in 1847, revived the operation of puncturing the cornea at its periphery to reduce the conicity. von Graefe[20] advocated removal of the epithelium and the application of a caustic. Bowman[21] attempted trephining near the cone, without good results. Bader[22] removed an elliptical piece of the cornea and allowed the lips of the wound to approximate. Badal[23] passed sutures thru the cornea before removing the apex. Critchett[24] removed an elliptical piece of the apex. Fox[25] performs an iridectomy first to prevent prolapse of the iris, which was a complication of Bader's[22] operation, and sutures the edges of the horizontal section to coaptate the margins of the wound, as suggested by Badal[23].

Gayet[26], in 1879, applied the cautery, producing perforation. Abadie[27], in 1887, advocated placing the cauterization area toward the periphery of the cornea, without penetrating deeper than Descemet's membrane. Andrew[28] used the cautery over the scarified area. Schwenk[29] reported permanent marked improvement of vision following repeated cauterization. Critchett[30] first applied the cautery at a black heat to the whole area to be scarred, destroying next a smaller area at increased heat, while the very apex was touched with the cautery at dull red heat. Tweedy[31] and Ziegler[32] pushed cauterization to the point of perforation. Elschnig[33] cauterizes the cone and then connects this area with the limbus by a bridge of burned epithelium, over which new vessels develop. He does not believe an iridectomy is necessary following this procedure.

In 1871, Albini[34] devised an aluminum plate to be held before the eye by the lids. His many claims for this device have never been realized, but many ingeneous arrangements have been attempted since to improve vision in cases of irregularity of the cornea. Small discs with stenopaic holes or slits can be adjusted by great care to improve the vision. The hydrodiascope of Lowenstein and the contact glasses[35] devised by Sulzer and Fick can be worn for short intervals with improvement of vision.

In most cases the vision can be improved to a greater or lesser extent by concave spheres and cylinders, but occasionally improvement is obtained by convex spheres and cylinders. Such a case I have had under observation for eighteen months.

N. M., age 15. Seen first in June, 1923, complaining of poor distant vision, but had never worn glasses. No pathology noted in cornea or fundus. Vision: R., 6/15; L., 6/45. Accepted under atropin:

R.+1.00⌒+2.00 Cyl. Ax. 180°, V=6/9.
L+1.00⌒+2.50 Cyl. Ax. 180°, V=6/15.

August, 1924, vision with correction: R., 6/9; L., 6/22. Slight irreg-

ularity of reflex in right cornea, but considerable change in left cornea without ulceration.

Accepted under homatropin:

R.+1.00⊂+1.50 Cyl. Ax. 180°, V.= 6/9+2.

L.+2.00⊂+2.50 Cyl. Ax. 165°, V.= 6/15.

Postcycloplegic:

R.+0.50⊂+1.50 Cyl. Ax. 180°, V.= 6/9+2.

L.+2.00⊂+2.50 Cyl. Ax. 165°, V.= 6/15.

General physical examination negative. Blood count normal. She is apparently perfectly well, and despite the poor vision is doing very fine work. Pilocarpin fails to contract either pupil, altho it has been used in sufficient quantity and strength to produce a violent headache.

In August, 1924, vision: R. E., 6/7—2; L. E., 6/15.

Occasionally the beginner finds in his rambles thru the literature of any subject a helpful bit of advice from one older and more experienced. I think Beard's[36] closing paragraph on this subject is of real help, when among other things he writes: "All things considered, the more conservative measures are the best, except for extreme cases. These consist in the prolonged use of miotics and pressure bandage. If no progress is made, repeated paracentesis of the cornea is subjoined, to which might, as an ulterior measure, be added the application of a small galvanic tip, in three or four short meridional lines, near the base. If these measures failed of material improvement, I should still advise against more drastic treatment, save where the ectasia is very great and the vision very poor, so poor, indeed as to hardly be called useful. True, much depends upon the condition of the fellow eye. If this be possessed of fair sight, one may venture further."

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BIBLIOGRAPHY.

1. Demours. Traite des Maladies des Yeux, Paris, 1818, v. I, p. 316.
2. Scarpa. Traite des Maladies des Yeux, Paris, 1802, v. II, p. 179.
3. Adams. Journal of Science and the Arts London, 1817, v. II, p. 403.
4. Schmidt, Carl. Abhandlung über die Hyperkeratosis, Inaug. Diss., Erlangen, 1830, p. 17.
5. Salzmann. von Graefe's Archiv f. Ophthalmologie, 1908, v. LXVII, p. 1.
6. Elschnig. Klin. Monats. für Augen., 1894, v. XXXII.
7. Sattler. A. J. O., Sept., 1898, p. 257.
8. Tweedy. Trans. Ophthal. Soc., U. K., 1892, v XII.
9. Imre. Szemeszet, 1922, v. LVI, p. 6. Zent. f. d. ges. Ophthal. u. i. Grenz., 1923, v. IX, p. 229.
10. Killick. Brit. Jour. Ophth., 1923, v. VII, pp. 264-269.
11. Fleischer. Klin. M. f. Augenh., 1922, v. LXVIII, pp. 41-50.
12. Gullstrand. Sven. Lakar. Handl., 1922, v. XLVIII, p. 103.
13. Pickford. Dublin Jour. of Med. Science, 1843, v. XXIV, p. 355.
14. Mackenzie. Practical Treatise of the Diseases of the Eye. Blanchard and Lee, Philadelphia, 1885, p. 658.
15. Adams. Journal of Science and the Arts, 1817, v. II, p. 402.
16. Tyrrell. Practical Work on Diseases of the Eye and Their Treatment, London, 1840, p. 277.
17. Critchett. Roy. Lond. Ophth. Hosp. Reports, 1859-60, v. II, p. 145.
18. Bowman. Roy. Lond. Ophth. Hosp. Reports 1859-60, v. II, p. 154.
19. Dix. Boston Med. and Surg. Journal, May 12, 1847, p. 289.
20. von Graefe, A. Archiv f. Ophth., 1886, v. XII.
21. Bowman. Ref. Am. Encv. of Ophth., 1914, v. IV, p. 2981.
22. Bader. Lancet, 1872. p. 73.
23. Badal. Archiv. d'Ophtal., Aug., 1910, p. 433.
24. Critchett. Practitioner, 1895, p. 426.
25. Fox. A. J. O., October, 1919.
26. Gayet. Lyon Médicale, 1879, v. XXX.
27. Abadie. Thése de Guiot, 1887.
28. Andrew. Brit. Med. Journal, 1884, v. II, p. 903.
29. Schwenk. Penn. Med. Journal, Aug., 1907.
30. Critchett. Practitioner, 1895, p. 426.
31. Tweedy. System of Diseases of the Eye, Norris and Oliver, v. II, p. 252.
32. Ziegler. Penn. Med. Journal, Aug., 1907.
33. Elschnig. Wiener klinische Rundschau, May 15, 1904.
34. Albini. Ref. Am. Ency. of Ophth., 1914, v. IV, p. 2995.
35. Dohme. Zeit. f. Augenh., 1922, v. XLVIII, p. 106.
36. Beard. Ophthalmic Surgery, P. Blakiston's Son & Co., Philadelphia, 1910, p. 388.

OUR KNOWLEDGE OF FOCAL INFECTIONS.

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A series of five cases here reported illustrates the relation of focal infection to iritis, chorioretinitis, hemorrhage into vitreous, abducens paralysis and glaucoma. The benefit of removal of such a cause is shown with the value of preventive medicine in the directions here indicated. Entrance Thesis submitted to the Chicago Ophthalmological Society.

When I ask myself this question: What, of the numerous discoveries or theories originated in medicine in the past two or three decades, will enable us to do the most good to mankind, I can think of but one answer. That answer is: The Conception of Focal Infection. This conception, while it did not arise over night or come from the brain of one man alone, being rather a product of the accumulated experience of medicine and medical men for years past, was first brought forward and kept in our attention by Dr. Frank Billings. The greatest part of the early experimental work was done by and under the direction of E. C. Rosenow. One man in this Society has for ten years been conducting distinguished experimental work in its application. To these men and many others we owe a large debt of gratitude.

At this point I have several case records to present, not because they bring out anything new, but so that your minds will be focused on this subject in a more definite manner, and because they are the kind of cases we are apt to see at any moment.

These are taken from among the records of my father and those of a friend, with whom I have discussed the subject.

Case 1. Mr. J. B. R. Young adult.

May 17,1900: Pain, redness of right eye; diagnosis, iritis; cleared up; no bad effects.

May 19, 1901: Rheumatic congestion of left eye; cleared up in several days.

May 25, 1904: Iritis left eye.

September 16, 1904: Iritis right eye.

1906: Iritis.

1908: Iritis.

May, 1912: Iritis, right eye; severe attack cyclitic spots; tonsils removed.

1913: Iritis twice. About this time he was found to have diabetes mellitus.

1916: Iritis.

1917: Iritis: Teeth X-rayed and infection cleared up.

No iritis since.

1922: Right vision, with correction, 6/5. Left vision with correction, 6/6.

Case 2. Mrs. L. R. Age 31 years.

May 29, 1924; two days ago, sight in left eye was noticed to be poor. It failed rapidly. Vision, R. E., 20/15—2. L. E., 3/200; fundi, right, normal; left, exudates in macula, recent. Diagnosis, acute left macular chorioretinitis.

Tonsils moderately enlarged. Throat never sore, but at times she would imagine sore throat was coming on. This would pass off in a few hours and she would forget it. Anterior pillars red, especially 4 or 5 mm. from margins; left most marked, and stippled. Purulent material readily exuding from the apex. Sinuses clear. Teeth O.K. when X-rayed.

May 30; urine negative; small doses of calomel started. Left vision, 2/200. Removal of tonsils advised.

May 31, tonsils out. On closing snare several drops of pus trickled out.

June 2, 1924: L. V.=12/200; Rx. protiodid of mercury, gr. 1/8, t.i.d.

June 17, 1924: L. V.=20/40.

July 16, 1924: L. V.=20/15 plus 4.

Case 3. Mr. C. N. G. Age 41 years. Occupation, weigher.

Says he has been totally blind in right eye since August 20th. All came on in one hour, with gradually increasing blur during that hour. Saw an eye doctor, who said he had ruptured a blood vessel and displaced the retina. Gave him no treatment.

Vision: R. E. Light perception and projection; L. E., 6/6.

Ophthalmoscopic examination; no red reflex. Olique illumination; great masses of grayish material moving about in the vitreous.

Diagnosis; hemorrhage into the vitreous.

Blood pressure; 123/80; Blood Wassermann negative; urine negative;

teeth, very bad condition, swollen gums, loose and decayed teeth.

Treatment atropin, hot applications, K. I. increasing doses, dionin powder. Bad teeth extracted. Patient seen but once a week on Sunday mornings; never stopped work.

December 3, 1922; counts fingers at ten feet.

January 14, 1923; right, vision 6/20 plus.

May 20, 1923; right, vision 6/10 plus.

December 14, 1924; no recurrence of hemorrhages; vision same.

Case 4. H. N. September 6, 1922. Paralysis left sixth nerve. Eye cannot be rotated externally beyond middle line. Had been in bed for three weeks with sore throat. Much prostration. Throat culture, staphylococci mainly—a few streptococci. Urine, amount very scant; albumin present, some blood, coarse granular casts, blood and amount variable. Mentally very clear. Note: Paralysis and kidney condition probably toxic—most likely will clear up.

Sept. 27: General condition greatly improved. Slight motion outward. Frosted glass over left eye.

Oct. 28, 1924. (Office) right eye, 42° prism blends the images. Fundi O. K. Vision with glasses 6/5 plus 3.

Dec. 9, 1924: Esophoria 5°, binocular vision; perfect motion.

Case 5. A. B., December 17, 1923; refracted; remains of iritic adhesions noted in left eye.

March 30, 1915; right iritis; right vision 6/15; left vision 6/6.

June 9, 1915; removed tonsils.

August 14, 1915; right iritis. During the following years his dentist was treating his teeth, and his occasional attacks of iritis continued up to 1922, when he had severe attacks in both eyes. Teeth X-rayed and infected areas relieved by extraction. Streptococcus viridans in culture. Rabbit injected—no iritis produced.

May 19, 1924; great pain in right eye previous night. Diagnosis; acute congestive glaucoma.

May 20, 1924; iridectomy.

Aug. 21, 1924; vision, R. E.=6/10 minus; L. E.=6/7 minus 2.

Present vision with glasses: R. E. 6/7 minus 1; L. E. 6/5 minus 1.

COMMENT.

Case 1. This patient has had over twenty attacks of iritis in a period of fifteen years, and says he had the same attacks as a boy. He has been free from this trouble for seven years, that is, since his dental infection was cleared up. He is a beneficiary of the focal infection conception. He also had diabetes. Some may ask if this may not have been a factor in the eye disease. I think it more probable that the iritis and diabetes were the result of one and the same cause—focal infection, probably of dental origin.

It is not an unwarranted stretch of the imagination to assume that the toxins from such a focus of infection might so effect the Islands of Langerhans in the pancreas as to prevent them from elaborating their internal secretion, necessary in the correct metabolism of the carbohydrates, or possibly the harmful effects on this organ were due to metastatic infection in its tissues. Possibly if we had possessed our present knowledge twenty years ago, these attacks might have been cut to one or two, and diabetes prevented.

The second case was one of acute macular chorioretinitis, which began to clear up after the removal of tonsils containing streptococci and pus. Protiodid of mercury was given later, but the improvement had begun with the tonsillectomy and there was no reason to suspect lues.

The third case was that of a man who had a hemorrhage into the vitreous, so severe that vision twelve weeks later was merely light perception and projection, but which cleared under ordinary local and eliminative measures, plus the removal of dental infection; so that vision seven and a half months later was 6/10 plus. No recurrence to date. The question of tuberculosis should have been investigated, but because he was seen but once a month, and wanted to keep working, this was not carried out.

The fourth case is one of left abducens paralysis and acute nephritis,

resulting from acute tonsillar infection and recovering after improvement of the latter.

The fifth case is one of recurrent iritis, finally yielding to the removal of infection about the root of a tooth. An added interest is the acute congestive glaucoma in an eye which had undergone several attacks of iritis.

In this series of cases there are several different eye diseases with one or two important sequellae, presumably the result of various forms of focal infection.

I have been informed that there are still some supposedly good eye men at large, who disregard the possibility of focal infection being the etiologic factor in iritis and other inflammatory eye diseases.

In 1915 and 1916, E. V. L. Brown and E. E. Irons, in their series of one hundred cases of iritis, found ninety-nine of them due to focal infection, and one in which the cause could not be found. Twenty-three of these were due to syphilis, nine to gonococcal infection, eight to tuberculosis, eighteen to dental infection, sixteen to tonsillar infection, three to sinus infection, three to genitourinary infection (nonvenereal), two to other infections, and seventeen to combined infections.

The differences between this series and their second series, published in 1923, does not bear directly on the subject as I am treating it here. However, it may be well to state that here also they found no other cause but focal infection, of course including syphilis and tuberculosis in this category.

I think enough work has been done and enough cases have been presented in the literature to make it safe to say that we are not treating the inflammatory and degenerative diseases of the eye rationally or thoroly until we have made a diligent search for and cleared up such foci of infection as are found to exist.

In treating cases similar to those cited, we must not forget the local measures and general eliminative procedures often so immediately necessary for the conservation of the eye and its vision, but nevertheless we should uncover the etiologic factory quickly, and eliminate it as soon as is consistent with the welfare of the patient.

Where does this lead us for the future? It leads to the subject of preventative medicine.

In Frederick Tice's Practice of Medicine, Joseph S. Evans says, "Both clinical and laboratory evidence at hand is sufficiently definite to permit the consideration of the following acute localized infectious processes as being due to hematogenous metastasis from primary focalized infection."

Acute endocarditis	Acute iridocyclitis
" pericarditis	" uveitis
" pleuritis	" episcleritis
" infective arthritis	" choroiditis
" tenovaginitis	" retinitis
" bursitis	" optic neuritis
Erythema nodosum	" thyroiditis
Purpura hemorrhagica	" pancreatitis
Acute bronchopneumonia	" nephritis
" cholecystitis	" myelitis
" enteritis	" poliomyelitis
" colitis	" meningitis
" gastric and duodenal ulcer	" osteomyelitis
" conjunctivitis	Herpes Zoster
	Myositis
" keratitis	Chorea
" iritis	Myocarditis

This knowledge, if rightly applied, should thru us very materially reduce the incidence of these acute diseases named above.

The focal infection conception also places in our hands the instrument for the prevention of a great part of the degenerative conditions of the eye, and will enable us to prevent, or delay for many years, the great degenerative conditions of the general system, which carry most of us off before our time. These are, broadly speaking: arteriosclerosis, chronic heart disease of various types, chronic nephritis and the host of maladies secondary to them, evidences of which we see with our ophthalmoscopes. I will not insult

your auditory centers with a list of these chronic diseases, but will leave that to your imagination.

We will turn for a moment to the ear:

A few months ago, Francis Emerson of Boston published an article in the Annals of Otology, Rhinology and Laryngology, in which he stated that many cases of chronic progressive deafness, most of them socalled nerve deafness, when treated by removing their foci of infection, not only held the hearing they then had, but actually gained in hearing ability.

He rather pooh-poohed inflation in these cases, in most of which he said the Eustachian tube was already wide open. This article at least possesses the merit of being rather refreshing to one who has seen many cases of chronic deafness inflated and reinflated, without any demonstrable improvement in hearing in a single case that he can recall.

This problem of prevention of focal infection, with its countless immediate and remote sequelae, begins at birth and comprises proper feeding, the correction of those congenital or acquired anatomic defects in the mouth, nose, pharynx, larynx, the gastrointestinal and genitourinary tracts which favor focal infection directly or by interference with normal function.

It includes the prompt removal of such foci of infection as do gain a foothold either in childhood or adult life. It includes especially in young and middle adult life a rational manner of living—sensible habits, eating and sleeping and daily outdoor exercise. I believe that some time ahead our working day will be so modified that every man will have an opportunity for daily open air recreation, and this is a change badly needed.

Looking at the problem in another light, it is a matter of cleanliness, and people should be gradually educated to the belief that cleanliness includes freedom from infection both open and hidden. The problem might be attacked by legislation, but I believe this is far from the method of choice. We now seem to be suffering from an overdose of law, regulation and standardization.

When we look into the future and see these diseases fading before our brilliant therapeusis as the mist before the morning sun, some may ask, Lo! what happens to the poor doctor in this sickless age? To allay your anxiety on this point I will borrow this from Dr. Frank Billings:

"The most optimistic of us cannot see that the application of tried and proved measures of disease and injury prevention, or the most hoped for correction of unhygienic conditions, or the greatest possible improvement of social life, will so materially diminish disease, morbidity or the incidence of injury to a degree that the medical profession will have nothing to do. Man is too immoral, or too careless, indifferent and selfish to permit a millenium of health to occur."

In closing, I express my gratitude to Dr. J. B. Loring and to my father for their valuable assistance and encouragement.

Chicago Ave and Jefferson Street.

BIBLIOGRAPHY.

1. Irons, E. E., and Brown, E. V. L. The Etiology of Iritis, J. A. M. A., 1916, v. LXVI, pp. 1840-1844.
2. Irons, E. E., and Brown, E. V. L. The Etiology of Iritis, Second Series, Sec. on Oph., A. M. A., 1923, pp. 263-283.
3. Evans, Joseph S. Practice of Medicine, Frederick Tice, v. I, p. 353.
4. Billings, Frank. Jour. of Iowa State Med. Society, 1922, v. XII, p. 40.
5. Emerson, Francis P. Is Chronic Progressive Deafness a Rhinologic or Otologic Factor? Annals of Otology, Rhinology and Larynology, 1924, v. XXIII, No. 3, pp. 865-890.

BILATERAL CONGENITAL MICROPHTHALMUS WITH CYST OF ORBIT.

VASILJ DERKAC, M. D.

ZAGREB, JUGOSLAVIA.

No eyeball could be felt, but in the cyst a solid body the size of a bean which cast a shadow by transillumination. Tapping the cysts yielded a yellow fluid highly albuminous. From the Eye Clinic of the University of Zagreb, Jugoslavia.

A female infant, 3 weeks old, was brought to the Clinic on May 27th, 1924.

The family is perfectly free from eye or other somatic malformations. The parents are in good health. The mother had one miscarriage. The patient is the sixth child, the fourth after an abortion. After birth the eyes began to discharge secretion which continues. The mother complains of the child's inability to open its eyes. The general condition shows no pathologic changes. The lids, especially the lower one, appear edematous, the skin is very thin bluish white, and prominent. Because of arching of the lower lids the lid fissure of both eyes is turned upward, arched and runs parallel with the eye brows. Opening the lid fissure we see the lower lids drawn upward, so that the upper border of the lower tarsus is looking downward while the lower one is turned upward, resembling the position of the upper tarsus in normal eyes.

The eyeballs are missing. The conjunctiva is inflamed, roughened, hypertrophied and covered with mucous secretion. Beneath the skin are palpable tender, fluctuating cysts, localized in the anterior part of the orbit and pushing forward the lower lid. No symptoms of increased tension in the skull on pressing the orbit, a sign that the cysts do not communicate with the cranial cavity. The cysts are movable. The right cyst is of the size of a pigeon egg, the left one is somewhat larger. At the temporal part of the left cyst we feel a solid formation of the size of a bean. On transillumination of the cysts by means of the diascleral lamp we see in the left cyst a thick, shadowy cloud, the size of a bean.

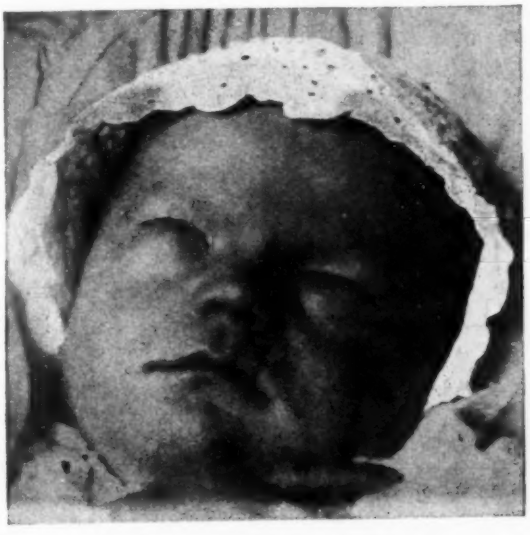

Fig. 1.—Case of bilateral congenital microphthalmos with cyst of orbit.

After 5 months the child was brought into the Clinic for the second time and we found two new bodies of the above mentioned size on the nasal side of both cysts. We punctured the contents of the cysts and found a yel-

lowish liquid, with 16.31 per cent of albumin in it. The content was bloody in its lower parts.

The above case is clinically definable as anophthalmus with orbitopalpebral cyst. The cysts are localized in the lower lids. The condition is the same on both sides. Similar cases have been observed by Lapersonne, Mitvalsky, Schimanovsky, Taylor, Collins and Natanson. This congenital malformation takes its origin in a faulty course of development in the earliest stage of fetal life. The normal succession of development mechanism is stopped, by some unknown causative factor, in the period of the primary vesicle of the eyeball. The vesicle does not sink in as is the rule in normal development, but continues growing at parts and becomes extended by accumulated liquid (Mitvalsky). The normal development of the eye is retarded, while the cyst becomes voluminous.

With the cell differentiation of the primary vesicle into the retinal elements, cells of the outer layer of the cyst wall (which is pushed inside in normal development forming the outer retinal layer, i. e., pigment epithelium) are now turned to the outside of the cyst; socalled *retina perversa.*

There is anatomically no anophthalmus in such cases, even if it is clinically right, because there is found a stunted globe, appended to and communicating with the large cyst. Nevertheless there are cases described of total absence of the eyeballs anatomically. (Hess, Fischel, Hanke, Van Duyse).

A yellowish, strongly albuminous liquid is contained in the cysts. Lens like remains and gliomatous vegetation may occur.

REFERENCES.

1. Bergmeister. Ein Beit. zur Genese der Orbitalzysten und der im Mikropthalmus vorkommenden Netzhaut and Aderhautanomalien. Graefes Arch., f. O., 1913, vol. LXXXV, p. 1.
2. Hippel, E. Graefe-Saemisch Textbook of O. Malformations of the Eye, 1900, p. 87.
3. Mitvalsky. Ueber die Orbital-Unterlidzysten mit Mikro— und Anophthalmus. Arch. f. A., 1892, vol. XXV, p. 218.
4. Natanson. Ueber Mikrophthalmus und Anophthalmus congenitus mit serösen Orbitopalpebralzysten. Graefes Arch. f. O., vol. LXVII, p. 185.
5. Peters. Die angeborenen Fehler und Erkrankungen des Auges, 1909, p. 59.

NOTES, CASES, INSTRUMENTS

VOLUNTARY NYSTAGMUS.

DANIEL B. KIRBY, M. D.

NEW YORK.

The ability to produce rapid voluntary nystagmus or ocular tremor in normal eyes is a quality that is possessed by few, if we judge by the small number of cases reported.[1,2,3]

The writer has observed two cases:

CASE 1. A girl of 12 years presented by Dr. Julius Wolff to the Ophthalmic Section of the New York Academy of Medicine on March 20, 1922. Thru the courtesy of Dr. Wolff, I am reporting this case. This patient was a deaf mute and desired eyeglasses with no apparent necessity for them. The vision and ocular muscle balance were perfectly normal, but at command or at will, she could start or stop rapid synchronous horizontal oscillations of the eyes. There could not be detected any involuntary nystagmus.

CASE 2. G. M., a boy of 18 years, a private patient of the author, mentally and physically normal, had as long as he could remember been able to make his eyes dance. His vision, accommodation and ocular muscle balance were normal. The central color vision and the fields for form and color were normal. Stereoscopic vision was normal. No scotomata could be detected on the Duane screen. The blind spots were normal. At will, he could initiate or stop a rapid horizontal tremor of both eyes. These movements were regular, rhythmic, synchronous and symmetric and in the horizontal plane. Analysis showed them to be alternations of equal components of eyes right and eyes left. Each ex-

cursion was about 5 or 6 mm. or about 20 degrees of arc, divided about the primary position. There was no involuntary nystagmus. Observation of the macula with the ophthalmoscope and of the anterior segment of the eye with the corneal microscope did not reveal any involuntary movements. A blurred image of moving objects in the visual path was obtained by the patient. He could not tell how he had discovered this faculty or how he accomplished it now. He could not produce a vertical nystagmus.

The only cases which are by their descriptions analogous to the author's are those of Benson, Lawson, Campbell, Gamble, Noyes, Dodd, J. W. Smith, A. E. Davis, Gowland and J. Wolff.

Dr. A. E. Davis has kindly written me that his patient, aged 32 years, whom he has known since she was a small child, is still able to bring about a voluntary nytagmus consisting of short, quick horizontal movements and lasting for 30 seconds or more. The vision and ocular muscles were entirely normal.

Dr. Alexander Duane has kindly written to me of the case reported by Gowland. The most interesting feature in Gowland's case was the considerable effort required to produce the nystagmus.

DISCUSSION. Nystagmus, as a term, signifies an involuntary oscillation of the eyes, so it is illogical to speak of a voluntary nystagmus. Rather call it a voluntary ocular tremor. Smith suggested the term ophthalmodonesis or voluntary tremulous or oscillatory movements of the eyes. It is generally accepted that visual sensation influences all the various movements of the eyes. Smith found that his patient could produce movements behind closed lids, or without looking at any object, and inferred from this that these ocular movements were not dependent upon afferent visual impulses, but on efferent stimuli from the corpora quadrigemina. Graefe stated that the individual possessing this faculty must at one time have had involuntary nystagmus, which has disappeared.

There was not in either of these cases any defect of vision, or any lesion of the ocular muscles, or of the nerves, or tracts, or centers controlling the muscles, that would necessitate an involuntary nystagmus either at the present or at any previous time. The mechanism of its production lies, probably, in the conscious control of the supranuclear centers governing the associated movements of the eyes in the lateral plane. These patients have learned to rapidly antagonize the associated movements of eyes right and eyes left, producing a tremor similar to that in other parts of the body, when muscles are rapidly alternately contracted as antagonists.

CONCLUSION. There exist certain individuals, who have the power to produce voluntary nystagmus or ocular tremor. This quality probably has no connection with the mechanism of true nystagmus. Its recognition might prove of interest in some medicolegal case.

30 W. 59th Street.

REFERENCES.

1. Posey and Spiller. The Eye and Nervous System, 1906, p. 279.
2. Duane-Fuchs. Ophthalmology Text Book, 1923, p. 343.
3. Pyle, W. L. Journ. Amer. Med. Assn., Dec. 5, 1908, p. 1940.
4. Wolff. Arch. of Ophth., Sept., 1922.
5. Gowland. Arch. de Oft. Hispano-Am., March, 1918.

NEUROPAPILLEDEMA IN CHLOROSIS.

G. N. BRAZEAU, M. D.

MILWAUKEE, WIS.

Chlorosis was first reported as a cause of Neuropapilledema by Bitsch in 1897. Since then, relatively few cases of this kind have been reported. In spite of many years of ophthalmic practice and after extensive reading of its literature I frankly confess that, prior to seeing the case which I will describe, I had never thought of chlorosis as a cause of neuropapilledema, nor had my able consultants. The best references that I could get were found in the French Encyclopedia of Ophthalmology. The ordinary textbooks

pass over this question entirely. The patient was a young girl of twenty. She had an attack of purpura ten years ago. She comes from a very neurotic family. Her sole complaint was that the sight in her right eye was entirely gone. A cursory physical examination of the girl showed that she weighed about ninety-eight pounds, was anemic looking, very nervous with choreic like movements in different parts of the body, appetite very finnicky and very easily fatigued on the slightest exertion. Blood examination showed hemoglobin 75%. Urine normal. No menstrual disturbances. This rapid examination of the patient was what led me to believe that chlorosis might be at the foundation of the case even tho brain or orbital tumors are the etiologic factors in the vast majority of the cases of neuropapilledema. The external appearances of the right eye were normal, tho the pupil was sluggish. The fundus showed a swollen and inflamed disc with its margins obscured by the mass of infiltration. There were no other changes in the fundus. The vision was reduced to bare perception of light. The left eye remained unaffected. The tentative diagnosis of a possible brain tumor having been made, consultation was requested. The diagnosis was confirmed. Not completely satisfied with our diagnosis in the absence of any other symptoms pointing to a brain involvement and remembering the patient's physical condition I advised giving the patient intravenous injections of cacodylate of soda combined with iron and to await results. Improvement both in the vision and in the general condition was at once apparent and in approximately ten days the vision had returned to normal. The diagnosis of chlorosis was confirmed.

While these cases are usually benign a certain proportion of them end disastrously by going on to optic atrophy and blindness. Whenever recovery takes place, it is generally much more protracted than in this case. How long it takes for an optic neuritis to develop is enigmatic, as a rule; here the development was complete in four days. These cases are generally found in severe cases of chlorosis, and yet the only symptom complained of in this case was the poor sight. The patient was able to carry on her college work without any apparent difficulty whatsoever. Why the eye lesion should confine itself to one eye is also a matter of conjecture. Anemia and chlorosis are usually considered synonymous. They are, however, very different, as the former is but a symptom while the latter is a definite morbid identity, the basic difference between them being their etiology. Chlorosis, in which heredity plays a large part, is essentially of nervous origin and is composed of both dyscrasic and nervous elements. Important as are the changes in the blood, nevertheless, they are secondary to influences of nervous origin. Chlorosis is itself fast disappearing as a disease (Moore, Emerson) and so are our chances of seeing its complication. Since a mild form of Bright's disease is often associated with this disease, may it not be that intoxication from the kidneys plays a certain role in the etiology of the papilledema, even tho there be no albumin present in the urine? The differential diagnosis between the eye lesions of anemia, chlorosis and Bright's disease may sometimes be very perplexing, as they bear such intimate resemblance to one another as to be almost indistinguishable. By far the commonest cause of neuropapilledema is brain tumor. This we should always remember when faced with an optic neuritis. It is very important that we determine its cause as delay might ensue which would necessarily compromise the chances of recovery. How long the treatment may be delayed without damage to the optic nerve in cases of chlorosis is also a matter of conditions and circumstances peculiar to each case. Cases like the above have undoubtedly been traced to sinus disease. I could not feel justified in exenterating sinuses in the absence of definite symptoms, as is being done of late.

700 Majestic Bldg.

A CASE OF BUPHTHALMUS.

JAMES ALBERT MORGAN, M. D.

HONOLULU, T. H.

Buphthalmia or hydrophthalmus is a disease characterized by a uniform, spherical bulging of the whole cornea. It generally takes the form of infantile or congenital glaucoma. The increase in size extends to the neighboring parts of the sclera and often involves the whole eyeball. It is always congenital.

The large size of the cornea is the most striking feature (megalocornea). The cornea may be opalescent, dull, bluish or it may be transparent. The anterior chamber is very deep, media clear. The blue color of the sclerotic, especially near the limbus is due to thinning. Sometimes there is nystagmus. The globe is much enlarged, usually oval and elongated, tho the spasticity of the young tunics is shown by an unusual increase in diameter of the corneal base.

Buphthalmus is quite different from most cases of glaucoma. But there is hypertension, a secondary cupping of the optic disc and also a chorioretinitis, the latter being often overlooked. Abadie believes that the chorioretinitis plays the fundamental rôle and directs his therapeutic measures against that disease. Miotics are of but little avail and no form of surgical interference can cure the disease. Iridectomy is not followed by particularly good results. Posterior sclerotomies, repeated, give the best results.

CASE. J. S., native Samoan. Age 10. The early history is rather obscure, as it was obtained thru an interpreter. There are six children in the family, this being the only one with eye trouble. It was first noticed about four years ago, that both corneas had a whitish appearance. The vision of the right eye began to fail about six months after the condition was noticed. Native herbs were used with no success and the eye commenced to shrink until at the end of four years a mere phthisis bulbi remains. The eyeball is very soft with an entirely opaque cornea and no light perception exists.

The left eye shows typical dark sclerotic with large cornea. The lens is opaque, making an examination of the fundus impossible. The patient is able to distinguish objects near at hand. The tension is slightly above normal.

The patient has been in the hospital for one month for observation. The blood Wasserman is strongly positive. To date, the patient has had four intravenous injections of sulpharsphenamin with potassium iodid. No operation has been performed, as the present vision is sufficient to allow the patient to take care of himself without help.

48 Young Building.

SCISSORS MAGNET EXTRACTION.

EDWARD JACKSON, M. D.

DENVER, COLORADO.

This operation is used to remove from the eye pieces of magnetizable steel, so held by exudates or scar tissue, that they cannot be drawn from their adhesions by the force of the magnet, even when the most powerful magnet available has been employed and the tip brought as near as possible to the foreign body. It was described in 1909. (Tr. Sec. on Ophth. A. M. A. p. 30).

To do this operation, the scissors are to be applied to the tip of the magnet, with the blades in the direction of the lines of magnetic force, and their slightly blunt tips as near as possible to the foreign body to be extracted. When the current is turned on the foreign body is pulled toward the magnet. As the tips of the blades are separated tissue is pushed between them, by the pull of the magnet on the foreign body. As the blades are closed some of this tissue is cut. When the blades are again opened more tissue is thrust between them, to be similarly divided.

By repeated snips of the scissors a tunnel is made to the foreign body, until it is in contact with the scissors tips. The snips being continued the foreign body is more and more separated from its bed, and comes more and more under the influence of the magnet; until, when

magnet and scissors are withdrawn together, the particle of steel comes away adhering to the tips.

The relations of magnet, scissors, lines of magnetic force and foreign body are shown in Figure 1. The scissors tips should be pushed to the resisting tissue,

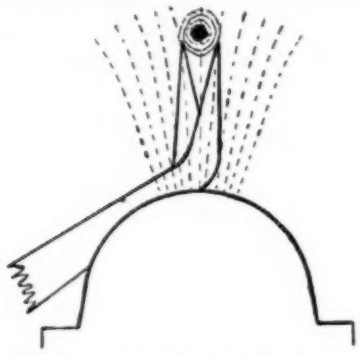

Fig. 1.—Diagram of end of magnet with lines of magnetic force and scissors blades brought close tc foreign body.

closed. Then, when they are separated it will be drawn between them. The snips should be wide enough to make a tunnel sufficiently large to carry the foreign body, and also to readily admit the scissors; but not unnecessarily wide. It is best to make a good many snips, 20 or 30 and, if possible, feel that the foreign body is in touch with or between the scissors blades before attempting to draw it out. During the snipping the scissors must be held steadily in one position.

If the first attempt is not successful the scissors may be slightly withdrawn (2 to 5 mm.), their exact direction carefully considered and readjusted slightly, to point more exactly toward the foreign body. Or the current may be turned off the magnet, and the readjustment of direction made. Then a second attempt similar to the first is to be made. This may be repeated 3 or 4 times. Of course it should be ascertained before attempting this, just where the foreign body is located and that it is magnetic. Accurate localization is necessary. Probably such foreign bodies are never of entirely recent lodgment. Nothing can be gained by hastening operation, before a satisfactory localization can be made.

The form of scissors deemed most suitable for this operation is shown in Fig. 2. The blades are bent to an angle of 60 degrees with the direction of the handles. This permits them to take the direction of the lines of magnetic force, when the joint is applied to the tip of the magnet, with the handles, in the position in which they are easiest to manipulate, without hiding the field of operation. The scissors are made by V. Mueller, of Chicago.

Fig. 2.—Method of holding scissors in contact with tip of magnet. A broader tip would be more powerful.

SOCIETY PROCEEDINGS

CONVENTION OF ENGLISH SPEAKING OPHTHALMOLOGICAL SOCIETIES.

This Convention, which was favored by large contingents from the United States, Canada, and the Netherlands, as well as several from the more distant Antipodes, was held at University College (University of London) on July 14th, 15th, 16th and 17th, under the presidency of Mr. E. Treacher Collins.

The work of the Convention was divided into two simultaneous sessions for the hearing and discussion of papers; while for two special events combined meetings took place. These events were a symposium on "The Evolution of Binocular Vision," and a debate on "The Microscopy of the Living Eye." The opening ceremony, the presentation of the Nettleship Prize, and the President's address took place in the Memorial Hall of the College.

Official Welcome.

The Rt. Hon. Neville Chamberlain, the Minister of Health, extended, on behalf of his Majesty's Government, a warm welcome to the delegates, especially those who had come from overseas. He said it had not been found possible yet to resume the international gatherings, owing to the backwash of the war, a circumstance he was sure was generally regretted. But those attending the Convention would find it conducted in a tongue that all could follow, and it had brought together peoples from the British Dominions and the United States, who were already united by such friendly ties. He referred with satisfaction to the great advances which had been made in ophthalmology during the past 70 years, and the efforts being made in this country, in the 482 ophthalmic clinics, to deal with and prevent eye disease, especially in the young. Under the presidency of Mr. Treacher Collins, whose name was so well known among ophthalmologists the world over, the Convention was bound to be a great success, and he believed members, when they departed, would be armed with a new vigor and confidence for the warfare on behalf of the most precious of the special senses.

His Excellency the American Ambassador, Mr. Houghton, expressed, on behalf of his American brethren, thanks for the welcome which had been extended to them, and to the British Committee for the arrangements made for their instruction and comfort. He then, in playful mood, deplored the fact that so many of the inhabitants of the States were being left without eye treatment while the ophthalmologists were disporting themselves in England, and uttered a word of warning as to the possible results of the feasting and entertainment provided.

Representatives of the various bodies composing the Convention were then separately presented to Mr. Chamberlain, who warmly greeted them.

The Nettleship Prize.

The President then presented to Prof. S. E. Whitnall, of Montreal, the Nettleship Prize (which is awarded triennially for the encouragement of scientific ophthalmic work) for his work on the anatomy of the orbit and accessory organs of vision. The recipient made a graceful acknowledgement.

Critchett Memorial Presidential Badge.

On behalf of a number of intimate friends and colleagues of the late Sir Anderson Critchett, Mr. W. T. Holmes Spicer invested the president with a handsome presidential badge, worked in gold and enamel, bearing the portraits of Sir Anderson and his father, George, and a Latin motto. Mr. Spicer recalled a number of personal characteristics and incidents concerning both, who, together, had practiced ophthalmology for nearly a hundred years.

Elimination of Eye Disease.

Mr. Treacher Collins, in his Presidential Address, said his purpose was to give some historic facts; which aided by imagination, might enable peeps to be obtained at some of the wonders which might some day be brought to pass. Work which had re-

cently been done made it possible now to contemplate the elimination of some eye diseases from the human organism.

In *leprosy* the eyes were frequently affected, and tho active measures had rendered it practically nonexistent in England, the Empire was said to contain 300,000 cases of the disease.

Dealing with *small-pox*, he said that about half the cases of indigent blindness applying for relief in this country in the last century lost their sight thru small-pox, but in 1922 a careful inquiry revealed only six cases due to the disease in the various institutions for the blind. Much of this improvement was due to the greater care of the eyes in all pyrexial diseases, and not only in cases of small-pox.

Ophthalmia was carried by an organism which did not produce spores, and as drying killed the organism, infection could only occur while the eyes had a moist discharge. Trachoma, the worst form of ophthalmia, had not had any one microorganism identified with it, but it was possible that here also the virus was killed by drying it, and measures based on this idea were largely responsible for the practical elimination of the disease from the British army and the Poor-law schools. At an inspection of Poor-law schools conducted by Edward Nettleship in London in 1874, 42% were found to have granular lids or trachoma, but in 1896 Sidney Stephenson could find only 4.91% of such sufferers.

Syphilis was held to be answerable for from 10% to 15% of the eye diseases of the country, but Mr. Collins considered that in the struggle against the spirochete now being waged man was bound eventually to win, especially after the "conspiracy of silence" concerning the disease was broken by the report of the Royal Commission on the subject. So serious were the consequences of syphilis that the President considered that when any person communicated it to another, wilfully, he should be incarcerated and treatment carried out until he was no longer infectious.

The influence of metastatic septic infection as a cause of *iritis* was now largely recognized, thanks to the convincing labors of William Lang, who found that in 37% of his cases of iritis, pyorrhea alveolaris was the only ascertainable malady.

With regard to *tuberculous eye disease*, Mr. Collins said he was not able to establish by figures that these were diminishing of late, but both in America and here the death rate from respiratory tuberculosis showed a gradual drop. He was able to speak, from a long experience of the Swanley Ophthalmia Schools, of the great benefit sufferers from tuberculous eye disease derived from an open air life, with good food and plenty of recreation.

Phlyctenular ophthalmia and its elimination was a social problem, closely associated with bad housing and congestion of the population, also with dietary errors, especially the absence of the fat soluble A vitamin.

He had something very definite to say on the prevalence of *myopia*, declaring that it was largely the outcome of our present system of education. It was unnatural to put the young growing animal to pore over books for several hours a day, often in artificial light; and he thought more use should be made by the education authorities of the cinema, the lantern, object lessons, and oral teaching. Lessons in geography, for instance, could take the form which would appeal to the young mind as a veritable voyage of discovery.

He made reference, in conclusion, to the work of the Council of British Ophthalmologists, the similar body in the United States, and the ophthalmic work of the League of Nations and the Red Cross Societies League.

Evolution of Binocular Vision.

At the symposium, largely attended, the chair was occupied by the president.

Professor Elliot Smith, F. R. S., said that anatomists could throw some light on certain aspects of this problem, or could at least define issues which others could more fully interpret. The fundamental fact which brought about the evolution of the primates was the cultivation of vision, and the increased reliance on it for

guidance. It led to the performance of a greater range of movements, some of them skilled, the development of which in man placed him in his position of supremacy. Fortunately, for the sake of this inquiry, in the case of the primates there were a series of creatures which had survived and escaped the devastating effect of extreme specialization.

The primates came into existence in North America, near Wyoming, before the beginning of the tertiary period, where they separated from the group of insectivorous animals. The retinae, cerebral cortex and oculomotor nuclei of these primates had been closely studied by Mr. Le Gros Clark in Borneo and other places. Shortly before the beginning of the tertiary period the lemurs came into existence, and in one class of them smell began to be replaced by vision as a guiding sense. The tarseus was a very important animal to study in this connection, as it had survived from the beginning of the tertiary period with practically no change.

The speaker was able, some years ago, without histologic technic, to map out the distribution of the cortical areas in a series of animals, and to show that, commencing with the jumping shrew, there was a gradual replacement of smell by vision, and since that time Mr. Le Gros Clark, by histologic methods, had confirmed the work. In the marmoset this supplanting of smell by sight was very marked in comparison with the tarseus. The chameleon illustrated in a striking way the prebinocular vision stage, as this animal could look in a forward direction with one eye, and, with the other, in a backward direction. A true macula lutea was found only in the apes and man.

Sir Frederick Mott, F. R. S., retraversed the ground he covered in his Bowman Lecture of nearly 20 years ago on the evolution of the cerebral cortex and binocular vision in mammals, contending that later researches had supported his thesis. Some slides he showed proved that sometimes the lunate sulcus, seen in all anthropoids, was occasionally met with in the human. With the rise of creatures in the scale, the peristriate area of the occipital lobe was seen to be increased, and the frontal region of the brain underwent increased development.

Professor Watson, of Baltimore, found that a baby could fix a light with its eyes soon after birth, but the hand movements in association with this fixation were not acquired until the 130th day of life. In the full term fetus there was a myelination of the optic radiations, and this was considered to be an agent in the fixing of the light. Sir Frederick contended that all our visual experiences were associated with tactile perceptual ones, and he showed a series of slides which illustrated work which Professor Sherrington and he had done together.

Prof. S. E. Whitnall (Montreal) said this problem had interested him chiefly from the point of view of the movements of eye muscles, during the gradual transition of the eyes, in succeeding species, from the side to the front of the head. Lemurs, and all animals below them in the evolutionary scale, moved their heads to see objects, instead of moving their eyes, and the extent to which human beings moved their heads was not appreciated until they suffered from a stiff neck. If in order to increase the visual field of an animal the eye was not moved, the orbit could be moved; while herbivora had an elongated pupil to increase the avenue of vision. He dealt in detail, by means of diagrams, with the changes in the visual relation to the median line of the head; and the alterations successively seen in pairs of ocular muscles. He said that many animals converged while feeding, but they did not possess stereoscopic vision.

Sir Arthur Keith, F. R. S., in view of what had been said by previous speakers, dealt almost solely with the main modifications in the skull and orbit in the process of reaching towards the acquisition of bifoveate vision, a term he preferred to binocular vision. Man probably took his human form in the Miocene period, but his ocular outfit had been acquired long before that time. He said that one of the greatest services rendered to anatomy was Prof.

Elliot Smith's clear proof that the evolution of bifoveate vision was the result of sight gradually replacing smell as the dominant guiding sense.

Sir Arthur thought this development was closely associated with the mode of climbing, for all bifoveate primates were jumpers, and they must be able to judge the distance of the branch they wished to reach. Some 24 years ago, Lindsay Johnson showed that monkeys, anthropoid apes and man had the same form of eye and retina and the same ocular muscle outfit. In ordinary monkeys head and eyes moved together, and the power of lateral movement of the eyes was small in them. In the great anthropoid apes conjugate movements were free and frequent, but convergence could not long be sustained. Man's superiority lay, not so much in the kind of eye with which he was furnished, as in the quality of brain behind the eyes. Still we lacked knowledge of the actual machinery which transformed the visual world as seen by the marmoset into that as seen by man.

On the proposition of Sir George Berry, M. P., seconded by Mr. J. B. Lawford, the speakers in the symposium were very cordially thanked.

Microscopy of the Living Eye.

On Friday morning the Sessions combined for a discussion on this subject, separate speakers dealing with different portions of the eye.

Dr. Gordon Byers (Montreal) spoke about the cornea. He said that tho the direct method of examination gave aids of great value, limits to its effectiveness were imposed by great difficulties of illumination. The value of the new method was chiefly in disclosing changes collectively, giving a meaning which could not be attached to isolated findings. Pathologic changes were now to be seen in the cornea with great distinctness, because of the variety of illumination now possible. Still, the slit lamp revelations were probably less important in regard to the cornea than in respect to some other parts of the eye.

Dr. Arthur Bedell (Albany, New York), who dealt with the vitreous, said the slit lamp offered a method for a more careful study of vitreous details than had previously been possible. In disease, the changed framework might consist in the massing of fibers and the liquefaction and absorption of structures, also in pigmentation. The slit lamp had made possible an exact prognosis in cases of penetrating injury; as it revealed the presence or absence of tension fibers, which might later cause a detachment. He showed a wealth of slides and much detailed information.

Mr. T. Harrison Butler (Birmingham) spoke of the lens. He said the retrolental space was a cleft filled with aqueous, and in cases of degeneration of the vitreous this disappeared. He showed drawings illustrating detachment in lens dislocation, in glass-blowers' cataract, and in an instance of shrunken lens. He said the lens sutures were known to constitute the sites of various kinds of cataract. The lens shagreen, he considered, must be due to the intimate structure of the capsule itself. He showed that an abnormal growth of the lens zones caused central cataracts and varied forms of zonular or lamellar cataract.

Mr. Basil Graves went into the optical properties of the new apparatus, and gave a number of illustrations by means of the epidiascope. He said it was desirable for the observer to train himself to observe the depth of the anterior chamber. The slit lamp should be so constructed, that the vertical width of the beam at its focal region could be made to cover the full diameter of the cornea and anterior chamber. The study of the aqueous fluid resolved itself into two factors: (a) observation of the outstanding beam, (b) observation of discrete particles.

Embryology of Lens.

Miss Ida C. Mann dealt with the embryology of the lens and the correlation of it with the slit lamp appearances. At the first stage she showed there could be seen the beginnings of the free cells, whose persistence were said by Vogt to be the cause of anterior axial embryonic cataract, which was named after that authority. But examination of this and succeeding stage-

showed this to be unlikely, as the opacities described in the adult lens were in the vicinity of the anterior Y suture, while the potential cavity of the lens vesicle was immediately under the anterior lens epithelium. She described the anterior vascular capsule of the lens when seen with the slit lamp.

Changes in Eyes Injured or Operated Upon.

Mr. T. Harrison Butler gave elaborate pictorial representations of some changes observed with the slit lamp in eyes which had either received an injury or had been operated upon. After a blow on the eye the lens was found to be displaced backward, as it was also after penetration by a steel fragment which was removed by a giant magnet. Glaucoma occurred some months after operation, in each case, and this glaucoma was cured by paracentesis. Radial ruptures of the iris were not discovered until the eye was examined by focal light. In three of his cases, examination with the slit lamp gave timely warning of an approaching sympathetic ophthalmitis, and prompt measures taken at this stage resulted in a complete recovery.

Conical Cornea.

Dr. L. Webster Fox (Philadelphia) presented a historic review of conical cornea and its surgical treatment, and described an improved operation for the condition. He said that occasionally after the operation glaucomatous symptoms arose, but only very rarely did a shred of iris become entangled in the wound. In one case there occurred opacity of the lens, but this was remedied by needling. His experience of the new operation in 40 cases had been very encouraging.

Intracapsular Extraction of Cataract.

Dr. A. H. Sinclair (Edinburgh) dealt with the subject of intracapsular extraction of cataract, and described the type of operation he had recently been carrying out with a new pattern lid retractor, double cross action forceps, and a new lens expressor and wound guard. When using the latter there had been no loss of vitreous in any of the 21 cases in which it had been employed. He gave a very interesting cinematograph demonstration of the actual operation. Where the lens was removed completely the average vision was 6/11, and where the lens was not removed complete in the grasp of the forceps it was 6/7.

Antigen Treatment of Cataract.

Dr. A. E. Davis (New York) contributed a paper on the lens antigen treatment of cataract, in which he contended that by the use of this antigen alone practically every case of cataract in its incipiency could be arrested, but mature senile cataracts he preferred to take away by surgery. The longest time during which he had had these cases under observation was four years, and the average loss of vision did not exceed 12%.

Muscle Recession for Squint.

Dr. P. Chalmers Jameson, of Brooklyn, placed before the convention the principles of muscle recession with scleral suturing, as applied for the correction of squint. He emphasized the importance of the site of the conjunctival incision; namely, at or about the curve of the semilunar fold. The muscle and the capsular flap should not be injured on the surface which was adjacent to the caruncle, and the scleral site should be carefully cleared of debris. He discussed the question at some length.

Pituitary Tumors.

Mr. Percy Sargent and Dr. E. C. Carmichael (London) dealt with the treatment of pituitary tumors; as they considered that the time had arrived when ophthalmologists should seriously consider what was to be gained by operation. They set out five classes of tumor, remarking that the adenomata were the most frequently occurring, and the most amenable to treatment. Moreover their diagnosis could usually be arrived at early. It usually manifested itself by visual disturbances, at first by an equal affection of both eyes, the visual loss involving first the upper and then the lower temporal quadrant. The authors discussed in detail the risks from attempts at removal by operation.

Cerebromacular Degeneration.

Dr. Gordon Holmes and Mr. Leslie Paton showed the ophthalmoscopic changes which occurred in cerebromacular degeneration of the Batten type. In most of the cases the retinal changes were fairly well marked when the patients came under observation. In very early cases there was seen a small whitish area at the macula, and this was surrounded by a reddened ring. At a later stage, wavy radiating white streaks could be seen on the retina, and later still there was dotting of the macula. In the third stage the evidences of retinal atrophy spread out to the periphery. Clinically the cases were closest allied to the group of amaurotic family idiocy.

Myopia.

Mr. Ernest Clarke (London) passed in review the various suggested causal factors in myopia, and said his view was in line with that of Sir Arthur Keith, that myopia was an inherited disorder which influenced the sclerotic at its early period. He threw out the suggestion that some extreme cases of myopia were a manifestation of a disease of the coats of the eye, myopia being a sequel and not a cause.

Preventative Measures.

Dr. Park Lewis, of Buffalo, discussed the scope and possibilities of preventative measures in ophthalmology, stressing the point that such work must be cooperative. The measures should include the prenatal treatment of interstitial keratitis, and early and thoro treatment of syphilitic pregnant women.

Teaching Ophthalmology.

Mr. Malcolm Hepburn (London) read a paper on the teaching of ophthalmology, contending that the teaching of the subject should vary in completeness with the different classes of students, which classes he placed into three categories: (1) undergraduates, (2) general practitioner postgraduates, (3) postgraduates intending to specialize in the subject. He criticized a number of practices now carried out, and formulated a scheme making for greater efficiency.

Maximal Lymphatic Reaction.

Dr. E. L. Jones, of Cumberland, U. S. A., read a note on the advantage of immediately producing a maximal physiologic lymphatic reaction, in eyes which had been dangerously wounded. He contended that lymph was Nature's chief agent for combatting infection and for repairing injured tissues. By injecting under the conjunctiva 2 c.c. of a 1 in 1,500 cyanid of mercury in the case of a wounded eye, one produced a great outpouring of lymph, and this had proved to be most beneficial, either in preventing infection, or checking it when it had commenced.

Milk Injections.

Mr. D. V. Giri (Eastbourne) contributed a paper on milk injections, and reported on a number of cases he had treated by the method. The result of these injections was, he said, to produce an intensive stimulation of the cells of the body as a whole; there was an increased formation of cells, and greater glandular activity. It was very valuable in hemorrhagic conditions, and in gonorrheal ophthalmia in the adult. Contraindications to its employment were severe traumatism, old degenerative processes, sympathetic ophthalmia, and atrophy of the globe. Iritis, whatever its cause, yielded in a satisfactory way to this treatment.

Future Conventions.

A general meeting of members of the Convention took place on Thursday afternoon to discuss the question whether the next meeting should be of an international character, and be arranged on a prewar basis. Dr. Howe proposed, Dr. Ring seconded, and the meeting adopted, the following resolution:

"That this Convention of English speaking ophthalmologic societies hereby empowers its president, Mr. E. Treacher Collins, to appoint a standing committee of five, of which he shall be chairman. The duty of this committee shall be to obtain such cooperation as is possible from representatives appointed by various national

ophthalmologic societies in promoting matters of international ophthalmologic interests, for example: (a) Aiding or initiating measures by international intercourse likely to prevent the dissemination of diseases of the eye and the lessening of hereditary blindness; (b) the establishment of uniform methods of recording results of visual tests and other matters pertaining thereto; (c) the interchange of ophthalmologic literature; (d) the promotion of international ophthalmologic congresses."

A further resolution, thanking the English organizers for what they had done, was cordially passed.

H. DICKINSON,
Reporter.

MINNESOTA ACADEMY OF OPHTHALMOLOGY.

APRIL 17, 1925.

DR. J. S. MACNIE, presiding.

Transient Exopthalmus With Acromegaly.

DR. J. A. WATSON (Minneapolis) presented the following case report:

Mr. C. K. T., age 45, consulted me on March 26th. He had been suffering with asthma for the past month and a half. His health had always been good but for several years past he had not been able to breathe freely thru his nose. He had, however, suffered from headaches all his life and there had always been a tendency to constipation. The blood pressure, heart and kidneys were reported normal by his attending physician. For some years he had been rather a heavy drinker, but had not touched liquor for the last 10 years. His general size and weight had increased so greatly and his features changed so much during the last 8 or 10 years of his life that relatives who had not seen him during that time were not able to recognize him. His weight had increased from 160 to about 240 pounds, tho in the last few months he lost about 30 pounds.

The nasal septum is very much thickened and deflected to the left with a large spur below. The upper part of the septum is in contact with the ethmoidal region on the left. There is very little illumination of the antrum or frontal sinuses, probably owing to the great thickening of the bones. There is apparently no purulent discharge into the nose. The mouth and tongue are greatly enlarged. The bones of the face are very large and heavy, especially the lower jaw bone. The size of this bone is best appreciated in the roentgenogram. All the sinuses are small and rather hazy, also probably on account of the great bone development. The sella turcica, however, can be seen to be greatly enlarged. The fingers are short, blunt, but not clubbed. The hands are short, heavy, thickened and spadelike.

The asthma has been greatly relieved by the use of adrenalin spray so that the patient is able to sleep comfortably at night. The breathing is quite quiet.

Dr. Watson said he would like to direct the attention of the Academy to the eyes of this patient. The fundus is apparently normal. The visual fields show only a slight concentric contraction. Occasionally one can observe a peculiar phenomenon apparently associated with certain eye movements, tho Dr. Watson said he had not been able to determine the exact movements that are most likely to produce it. It consists in the extrusion of the eyeballs, almost amounting to a forward dislocation. At such times the forward movement can be very clearly seen. It is so marked that one can readily grasp the eyeball in his fingers, but it always returns to its place in the course of two or three seconds. It seems to be an entirely involuntary movement.

Dr. Watson asked for the opinion of the Academy as to the advisability of operative interference on the nose for the purpose of relieving the asthma, or on the pituitary for the purpose of influencing the course of the disease.

Discussion. DR. FULTON asked how long this had been progressing. Dr. Watson stated that the history is indefinite, but about 15 years. Dr. Fulton asked if the patient had received

any treatment, and Dr. Watson stated that he had not as the case was never diagnosed.

Dr. E. J. Brown stated that about 30 years ago, in New York City, one of the doctors took him in to see an old woman with acromegaly. The woman's head was so large that it looked almost like the head of a horse.

Dr. Watson said that one of the peculiar features about this case is that there should be such a marked enlargement of the sella, without any impairment of the visual fields. He was unable to explain the extrusion of the eyes, and stated that he had never seen anything like it.

Blindness Following Inhalation of Carbon Monoxid. Retina Detached.

Dr. Wm. R. Murray (Minneapolis) presented a patient, male, age 24, farmer, who entered the University Hospital February 10, 1925, on account of impaired vision in right and left eyes of two weeks' duration. On the day previous to the onset of blurred vision he was exposed to the exhaust gas of a gasoline motor for two hours, while working in a garage, and gave a history of similar exposures on several previous occasions. The onset of the blurred vision in the right eye was accompanied by the appearance of a yellow spot, which later changed to a darker blurred area in the field of vision. Two days after the appearance of the yellow spot in the right one, the left eye became similarly affected, a yellow spot appearing, which likewise became darker. In both eyes the blurred area appeared to have a revolving yellow halo around the periphery. He had diplopia at times, some nausea and vomiting, and headache.

On the day preceding admittance, he was registered at the out-patient department, where he was seen by Dr. McKinley and Dr. Goss. Examination of the eyes at that time showed: slight tenderness of the eyeballs on palpation, normal tension, injection of bulbar conjunctivae, inequality of pupils, reaction to light and convergence present, intraocular media clear, disc margins blurred, retinal arteries slightly contracted, and vein relatively engorged. Visual fields showed contraction with sector defects, for red and blue; no scotomata present and no enlargement of the blind spot. He was unable at this examination or at subsequent ones to distinguish green color. Vision in the right eye was 20/200; left eye, 10/200.

On admittance to the hospital a general physical examination showed lungs, heart, kidneys and abdomen negative. Neurologic examination was also negative. Blood analysis: urea nitrogen 12.13; creatinin 1.80; sugar 0.099; Hb. 97; red blood cells 4,700,000; white blood cells 8,200; polymorphonuclears 60%; lymphocytes 36%; large mononuclears 2%; transitionals 2%. Blood Wassermann was negative. Spinal fluid: Clear with normal pressure; cell count 214; Nonne +; Noguchi +; Wassermann negative. Carbon monoxid hemoglobin with NaOH test was positive. Two days later spectroscopic test for carbon monoxid hemoglobin was negative, and NaOH test for carbon monoxid hemoglobin was also negative.

Ocular examination: Bulbar and palpebral conjunctivae mildly injected; extraocular muscles normal; no ptosis and no nystagmus. Pupils slightly irregular; right pupil larger than left with sluggish reaction to light and convergence. Intraocular media clear. There was bilateral papilledema, contraction of retinal arteries, and dilated veins. The retina presented a diffuse, grey-white opacity which extended uniformly thruout the fundus and was but slightly elevated; there was a subretinal edema present. Vision was failing rapidly and he was able to distinguish moving objects but was unable to recognize individuals. When he left the hospital one week later his vision was reduced to the perception of hand movements before right and left eyes. This patient later came under the observation of Dr. Burch of St. Paul, who reports a total bilateral retinal detachment. (Lantern slide illustration of fundus picture.)

Discussion. Dr. E. J. Brown was inclined to think that it is rather surprising that there are so few cases of carbon monoxid poisoning, as many

use gas ranges and are not careful about ventilation; some have no ventilation and are used, especially in the winter, with closed doors and windows. He stated that a young colored boy died from poisoning of that kind just a few weeks ago, about a block from Dr. Brown's home. The mother went away, leaving a woman doing the washing, and this young boy in the house. When she returned some hours later she found the boy lying on the floor dead, the woman sitting in a chair unconscious, the doors closed, and the gas range going full head. Dr. Brown stated he was called last Saturday to a woman who had an acute frontal sinus affection. He found they were using a gas range that had no ventilation. She told him that every time she used the gas range she had a headache and got very weak. Dr. Brown thought something ought to be done, legally, to bring this under the control of the Public Health Department.

Dr. Grant stated that when he saw this patient about 3 weeks ago his right eye had a moderate ciliary injection. There may possibly have been some conjunctivitis but there was nothing to account for it. In the right eye the cornea was clear, pupil fixed, lens slightly hazy and mottled thruout. The fundus gave no red reflex but appeared as if looking at a white wall. The left pupil was 5 mm., cornea clear, pupil fixed, no fundus reflex, tension full but normal. We were able to dilate the pupil of the left eye to some extent with atropin, at which time the cornea still remained clear, and the iris slightly hazy. At the present time the pupils of both eyes are in middilation and fixed, lens is distinctly mottled and one is able to get a white reflex thruout the entire fundus.

Dr. Burch said he was able to determine a detachment. Dr. Grant stated that he could not determine whether it was a detachment or exudate, altho there was a distinct elevation just posterior to the ciliary region, but no vessels can be made out at any point. For this reason he did not see how one could be absolutely sure of detachment. He also said that in detachment the tension would be much lower than it is at present. The exudate appeared to him to be in front of the retina.

Dr. Grant stated that, coincident with this, there were two patients in the hospital at the same time, both with retinal hemorrhages of undetermined origin. One man was in for the 3rd or 4th time. Both of these men were garage workers and had no physical signs or symptoms to account for the retinal hemorrhages. In the first case the patient had a very extensive hemorrhage in the left eye 3 or 4 years previously. This cleared up and left a typical picture of retinitis proliferans. The fundus became clear. New vessels formed on the exudate and hemorrhages recurred from the new formed vessels. Since that time he has had recurrent hemorrhages of the right eye. This patient is a very robust man, weighs about 200 pounds and is the picture of health. There is nothing in his general physical condition, so far as can be determined, to account for these lesions. The other patient was a very similar case. Vision had been lost in the right eye by an accident several years before. This man had a practically normal fundus, with the exception of scattered punctate and flame shaped hemorrhages thruout the retina.

Dr. Grant stated that both of these cases occurred in winter and it seemed possible to him that while working indoors they might inhale enough of the gas to weaken the vessel walls. The picture did not seem consistent with tuberculosis of the retinal vessels. The only symptom present was a vague headache thruout the vertex. These hemorrhages always clear up. He stated that he had seen the first case mentioned just a few days ago, and that the condition was entirely normal again. Probably in a short time the man would have other hemorrhages. It occurred to Dr. Grant that perhaps these men absorbed enough carbon monoxid to have some effect on the retinal vessels. At least, there was no other cause to satisfactorily explain the retinal hemorrhages in their cases.

Dr. Berrisford said he thought that the subject was important from a

medicolegal standpoint, with special reference to the Workmen's Compensation Act. It is a well recognized fact that there are certain young individuals in whom intraocular hemorrhage occurs, of which, despite, our utmost scrutiny, we are unable to determine the true etiology. If then such a hemorrhage takes place in a garage worker, where, during his employment, especially in winter, carbon monoxid poisoning is not at all uncommon, are we to attribute this lesion to carbon monoxid poisoning? Carbon monoxid poisoning is exceedingly common in garage workers; relatively speaking, in such cases intraocular hemorrhage is rare, at least in the light of our present experience. It would appear, then, that where an intraocular hemorrhage is discovered in a garage worker, the condition may be, in some instances at least, coincident with such employment rather than the result of it.

Dr. J. S. Macnie (Minneapolis) reported the following case:

Mr. G. B., age 40, reports that he was quite well up to 5 years ago. He was operated for hernia 5 years ago and never seemed to pick up. Following this he had a lame knee for 2 years. In 1921 he had had a bad abscessed tooth treated and several other diseased teeth pulled. A year following this the knee recovered and has never bothered him since. He has been married 21 years, and has 6 children, all well. For 4 years he has been under severe strain because of an invalid wife, necessitating, in addition to his own work, his taking care of the house and taking care of 6 small children.

His complaint now is that for 3 years he has had a progressive loss of energy. He sleeps fairly well but wakes up feeling tired and for the last 3 years has had severe headaches. His head aches all the time, and is especially severe when he wakes up at night. These headaches he describes as being all thru the top of his head. He has had several smothering spells and feels short of breath. These spells are transient and he has never lost consciousness with them. His appetite is good; drinks 8 or 10 cups of coffee a day; smokes considerably—cigars, cigarettes and a pipe. His physician states that the heart, lungs and kidneys are normal. The blood pressure is 100 over 60. The blood examination shows 80% Hb.; erythrocytes 5,040,000; leucocytes 9,750; P.M.N. 49; small lymphocytes 43; large lymphocytes 6; eosinophiles 2. His report is negative as to other blood findings. The blood urea nitrogen is 12.38 mgms. per 100 c.c.

This patient was sent in to be refracted, thinking that his headaches might be due to eyestrain. He has in the right eye a $-1.25\bigcirc-.75$ ax. 180°; in the left eye a $-1.25\bigcirc-.25$ ax. 110°, which gives him 20/20 vision in each eye. The ophthalmoscopic examination shows in the right eye O.D. margins clear, deep physiologic cup, veins considerably enlarged with marked pulsation; arteries show accentuated white lines and strap veins, somewhat tortuous. The general appearance of the fundus is mottled. The left O.D. —the upper inner aspect shows a clear outline. The disc as a whole is somewhat congested. The veins are markedly congested and in the inner choroid show marked tortuousity and are twisted. Pulsation marked. Near the macula there is one comparatively large flame shaped hemorrhage and between this and the optic disc are several small, mostly recent, and a few old, hemorrhages. The fields are markedly constricted.

X-ray shows the left antrum slightly cloudy. The sella seems somewhat enlarged.

Discussion. Dr. Murray asked if blood studies had been made. Dr. Macnie stated that the blood studies were negative in every respect. Two Wassermanns had been negative. There was no blood dyscrasia so far as he was able to report.

Dr. Fulton asked if it was not possible that this is a common case of thrombosis of the retinal veins; the walls of the veins simply being in a dilated condition. He asked if the teeth had been examined. Dr. Macnie stated that several teeth had been extracted and the others had recently been pronounced all

right. There was some cloudiness of the left antrum.

Dr. Fulton asked if the condition is the same on both sides. Mr. Macnie said it is more pronounced on the left side.

Dr. Brown asked if it might not be due to tobacco or caffein. Dr. Macnie stated that it might be; that the man is a heavy coffee drinker. Dr. Macnie asked if any one had ever seen that produce such an appearance in the fundus. He stated that the condition, taken in conjunction with the general history, is rather puzzling.

Walter E. Camp, M. D.,
Recorder.

COLORADO OPHTHALMOLOGICAL SOCIETY.

April 18, 1925.

Dr. W. C. Finnoff, presiding.

Tansley-Hunt Ptosis Operation.

F. R. Spencer and C. L. La Rue, Boulder, presented a man aged thirty-six years, who, eight years previously, had been operated upon for bilateral congenital ptosis by the Tansley-Hunt method. The upper lids covered the upper third of the pupillary area of the cornea, but it was doubtful whether proper closure of the lids could have been obtained if the surgical intervention had been more extensive.

Central Retinal Exudates.

W. C. Finnoff, Denver, presented a man, aged twenty-seven years, who had come on account of partial failure of vision, which had begun about a year and a half previously with a distorted appearance of images seen with the right eye. No distortion had been noted with the left eye. The patient had a generalized inflammatory rheumatism in 1912, and had been almost frozen to death a month before he had first noted failure of vision. The tonsils had been removed in September, 1924. Other examinations were negative, including teeth and sinuses, and spinal and blood Wassermann. At each posterior pole, scatered over a radius corresponding to three disc diameters, were numerous white areas in the deeper portion of the retina, a few of them bordered with a faint ring of pigment, and some having a dirty brownish center. In several places the areas were elongated and located under the retinal vessels. The macular region was peppered with very fine brown pigment dots. There had been no change in the appearance of the fundus since the patient had been first seen on December 6, 1924.

Discussion. W. H. Crisp, Denver, thought it not impossible, that the extreme chilling to which the patient had been subjected might have been responsible for the eye condition, by nutritional disturbances resulting from circulatory changes.

Tumor in Anterior Choroid.

W. C. Finnoff, Denver, presented a man, aged forty-one years, who had come on account of recent disturbance of vision of the left eye, the vision of the right eye having been lost a number of years previously. The cause of the blindness of the right eye was a simple optic atrophy. External examination of the left eye showed engorgement of the anterior ciliary veins in the uper nasal quadrant, and flattening of the upper nasal quadrant of the pupil when dilated. There were a few membranous floaters in the anterior vitreous. The retina in the whole upper nasal quadrant of the fundus was detached, and came forward as much as twelve or more diopters in the peripheral portion. When the eye was rotated upward and to the right, a salmon colored mass was visible in front of the detached retina, and the vessels in the mass were irregular in arrangement, and differed strikingly from the retinal vessels. The mass could be readily seen by focal illumination when the right eye was directed upward and to the right. With transillumination a distinct shadow was present in the pupil as the light passed over the area corresponding to the mass, and with transillumination from the lower temporal side of the globe a definite shadow was cast on the sclera in the upper nasal quadrant.

Exophthalmus from Ethmoiditis.

W. H. Crisp, Denver, reported a case of exophthalmus with pronounced edema

of the eyelids, in a child of eighteen months, apparently due to ethmoiditis. Less than a week before the onset of the eye disturbance, there had been an infection of the right ear without an apparent cold. Swelling of the right eyelids developed rapidly, in the course of twenty-four hours, there being a red edema of the lids which extended to the orbital margin. The lids were easily opened for examination, and the eyeball appeared absolutely normal, without chemosis or redness of the bulbar conjunctiva, altho twenty-four hours later there was a very slight watery appearance of the bulbar conjunctiva. Within twenty-four hours after the eye disturbance was first noticed, there was proptosis of about 3 mm. The temperature was at that time 103° by rectum, and the child was bright and did not appear decidedly ill. There was doubtful slight tenderness to pressure on the eyelid. The case had been diagnosed as probably one of ethmoiditis with edema of the deep orbital tissues, perhaps purely toxic in character, or perhaps associated with a bulging of the lamina papyracea. Next day, under internal administration of salicylates, the temperature was fairly steady around 102° rectal, and the lid edema not greater but perhaps slightly less. There was no distention of the retinal veins. Again a day later, the temperature was about 100° rectal, and the general appearance of the child much better. There was a marked shrinkage of the lid edema and free discharge from the nose, altho the proptosis was still quite marked.

WM. H. CRISP,
Secretary.

ST. LOUIS OPHTHALMIC SOCIETY.

FEBRUARY 27, 1925.

DR. JOSEPH W. CHARLES, presiding.

Hydrophthalmus.

DR. H. D. LAMB read a paper, giving a clinical report and microscopic findings in a case (see p. 784, this issue.)

Ciliary Ganglion.

DR. SHAHAN contributed a discussion on the human and comparative anatomy of the ciliary ganglion and reviewed the controversies on its nature and function, as recorded in the literature of the past seventy-five years.

Angioneurotic Edema, Quincke's Disease.

DR. J. F. SHOEMAKER reported the case. A. H., female, 12 years of age, was brought by her mother, January 9, 1925. The mother stated that when the child arose from sleep that morning the lids of the right eye were swollen. There was no pain, itching, or inflammation. She had had several similar attacks during the past summer and autumn, lasting about a day at a time. The patient was apparently entirely healthy, not complaining of any indisposition.

On examination there was found present a marked swelling of the lower lid of the right eye, which extended down over the malar bone. There was no sign of any inflammation of the skin and but the mildest form of conjunctivitis. Otherwise she seemed perfectly normal. She was given a mild collyrium to use at home and by the next day the edema had disappeared.

Quincke's edema or angioneurotic edema are different names applied to ephemeral spots of edema which may appear in the skin on any part of the body, in the mucous membrane or even in the synovial membrane. These spots of edema frequently resemble urticarial spots, and Osler said they were "only urticarial wheals writ large." The onset is sudden, there is no inflammation or local pain, and the condition disappears as quickly as it comes.

Etiologically the edema was first thought to be due to a neurotic factor. Later it was thought that it might be due to sinus infection. But the past few years the tendency is to associate it with a congenital hypersensitiveness (allergy), or an artificial sensitization to a foreign protein (anaphylaxis). Further investigation seems necessary before its true cause can be definitely assigned.

Convergent Squint.

DR. J. F. SHOEMAKER reported the case of a baby, 5 months old, who was

brought by her mother, October 31, 1924, with the following history:

When ten weeks of age she got a small piece of lime in the right eye which the mother removed at once. The eye was slightly inflamed for several days, but soon cleared up and was apparently all right. Two weeks after this the right eye began to turn in. The mother, thinking the trouble might disappear, waited until the baby was five months old, before seeking advice. During the two months' time the eye remained constantly crossed.

On examination it was found that the right eye was markedly turned in, it being in line with the left eye on looking to the extreme left, but was crossed when looking straight in front of her, and never turned outward past the median line. Otherwise the eyes appeared perfectly normal. Retinoscopy was very unsatisfactory but she apparently had between 3 and 3.50 diopters of hypermetropia. She had always been perfectly healthy.

Atropin drops were used in the left eye, and later a bandage applied over this eye, hoping to increase the motility of the crossed eye by compelling its use. This treatment had no effect, apparently, on the affected eye; so it was stopped. However, after several months the eye gradually turned past the median line at times and when last seen could be turned outward, possibly 15 or 20 degrees beyond this line, there being much better motility.

Discussion. Dr. John Green, Jr., gave two possible explanations. First, that there may have been some birth injury of the external rectus. (Was inquiry made as to prolonged or instrumental delivery?) Second, there may be an absence of the external rectus or a replacement of this muscle by an inextensible fibrous band, whereby abduction is impossible and abduction is often accompanied by retraction of the globe.

Dr. Jos. W. Charles: Dr. Green has mentioned the two possibilities I have in mind. It is quite feasible to operate in some of these cases at any rate. In 1910, a child 27 months old was brought to me for left converging strabismus and given plus 1.25 Sph. R. and L. In 1912 I prescribed +2. Sph. ⌒ +1 cye. ax. 90°. With this glass R. V. = 19/30; L. V. = 19/120. Constant occlusion of the right eye, and atropia at home did not improve vision; and in 1914 a diagnosis of congenital weakness of both externi was made.

In 1917 a very small but undoubted defect of the macula of the left eye was discovered. Tenotomy of the left internus was performed in 1920 after which it was found that an advancement was not needed. She had discarded her glasses. R. Em. V. 23/15. L., fingers at 2 feet.

Dr. J. F. Shoemaker (closing): Several things argue against this case belonging to that group of cases where there is an absence of the external rectus. In the first place both eyes were apparently straight the first three months of the baby's life; and secondly, the improvement that has taken place after two or three months. Both of these facts seem to prove the presence of the external rectus muscle.

THE NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

March 16, 1925.

Chairman, Dr. Robert Sullivan.

Eye Injury.

Dr. E. B. Cayce presented the case of W. D. F., a white male 22 years of age, who was first seen on the night of February 10, 1925, and gave the following history: Was cutting a button off a pair of overalls and the scissors slipped and cut the right eye. Examination showed a large cut extending entirely thru the cornea, with iris protruding thru the wound, which extended thru the sclerocorneal junction above. Protruding iris was clipped and he was given an injection of cyanid of mercury. The eye healed kindly and the absorption of the lens was as rapid as usual.

On March 14th, the patient returned with the eye irritable and a rather marked circumcorneal injection. 6 cc.

of sterilized milk were given in the buttocks. This controlled the irritation and the patient returned to work in four days following the injection.

Discussion. Dr. W. G. Kennon thought Dr. Cayce fortunate in that the man's inflammation subsided, but he believes it will come up again and that the eye will have to be enucleated.

Dr. M. M. Cullom agreed with Dr. Kennon. This eye presents a very dangerous appearance and he believed it should be immediately removed.

Dr. Robert Sullivan would like to know how many used aolan in preference to sterile milk, and what was the opinion regarding it in comparison with the latter. Personally he used the aolan as it is easier to get, easier to handle, and gives much less reaction.

Fred E. Hasty, Editor.

BALTIMORE MEDICAL SOCIETY SECTION ON OPHTHALMOLOGY.

April 7, 1925.

Simultaneous Cerebral and Ocular Vascular Lesions.

Dr. Harry Friedenwald read a paper on this subject.

Coloboma of Mesodermal Layer of Iris.

Dr. Jonas Friedenwald read this paper.

Conjugate Upward Movements of the Eyes, Postencephalitic.

Dr. Leslie B. Hohman reported four cases in which from one to six years after an attack of encephalitis, the patients developed recurrent attacks in which their eyes were turned upwards. The attacks last from a few moments to several hours. During the attack the patient is unable to turn his eyes down. Cases of a similar kind have been reported by three German authors during the past year. All the patients had more or less marked Parkinson's syndrome.

Discussion. Dr. Jonas Friedenwald stated that he had the privilege of seeing three of Dr. Hohman's cases. None of these showed any ocular troubles whatsoever during the intervals between the attacks. Ocular movements were well performed, in all directions, and muscle balance was normal.

Dr. Alan Woods asked why the condition might not be considered a paralysis rather than a spasm?

Dr. Harry Friedenwald said that before the encephalitic epidemic he had never seen such cases, tho they have been described in text books as hysterical manifestations. Six cases of this kind had been reported by Wilbrand and Saenger as hysterical.

Dr. Hiram Woods said he had seen a case in which an upward spasm could be produced by the patient's trying to look slightly above the horizontal line. The spasm would last for a few moments, after which the eyes return to normal position. This symptom preceded by some weeks the development of a typical encephalitis. After recovery from the acute illness, the patient has these attacks less and less frequently, for some time.

Dr. Hohman in closing the discussion, stated that he believed that the symptoms were due to spasm rather than paralysis, because the movements were conjugate, often associated with spastic movements of the head, and because no weakness of the ocular movements could be detected between the attacks. He believed that these cases differed from the ordinary hysterical ones, because they were not affected by psychiatric therapy and because cases of hysteria like symptoms, developing after encephalitis and unamenable to psychiatric therapy, have occurred in large number. He believed that these cases are due to definite organic lesions of the brain, but the localization of these lesions is as yet obscure.

Jonas S. Friedenwald, Secretary.

CHICAGO OPHTHALMOLOGICAL SOCIETY.

FEBRUARY 16, 1925.

DR. C. P. SMALL, President.

Melanosarcoma of Choroid.

DR. FRANK BRAWLEY presented and reported a case. The interesting feature was that after two years there had been no evidence of recurrence. The case had been treated with radium, which accounted for the loss of the cilia. (Reported in full p. 790).

Discussion. DR. WESLEY H. PECK believed that the tendency was almost invariably toward metastasis. Several cases he had observed had developed metastasis within three months, mostly of the liver, and he had never known any to live more than six months. This case argued very strongly for the use of radium following enucleation to prevent recurrence.

DR. HARRY GRADLE asked what were the definite indications for the use of radium?

DR. BRAWLEY said that it was done purely as a precautionary measure. The specimen from this case had been presented at the staff meeting at St. Luke's Hospital, and the surgical staff were very pessimistic as to the outcome. They said there was practically never a case without metastasis, which was fatal.

Obliterating Endarteritis.

DR. HALLARD BEARD presented a case in which lesions could be seen with ordinary light in the central part of the fundi, consisting of hemorrhages from small arteries, white streaks, the remains of obliterated arterioles, and extreme tortuousity and varicosity of the smaller veins. A segmentation of the blood column in the branch of the central artery which ran toward the fovea centralis, could be seen with red free light. The (stationary) blood column showed an inky black with red free light, while it was not visible by ordinary light.

Exophthalmos as a Complication of Nasal Sinusitis.

DR. C. F. YERGER read a paper on this subject which will be published.

Discussion. DR. FRANK BRAWLEY believed that too much value had been placed on X-ray diagnosis, possibly thru the enthusiasm of the X-ray men. One case he had in mind showed an absolutely negative X-ray report, and when the case came to operation, it showed complete destruction of the inner third of the supraorbital ridge and loss of a large area of the posterior frontal sinus wall. There was a thick mass of fibrous connective tissue covering the defect in the sinus wall. Strangely enough, there was no damage to the eye, no exophthalmos, and this in spite of the fact that there was a pocket extending into the orbit containing two grams of very thick pus. The patient made an uneventful recovery without any damage to the eye. The only explanation he could offer for the negative radiogram, was that the destroyed bone areas made a normal shadow possible. This X-ray work had been done by one of the best men in town, and when he heard what was found on operation and viewed the picture again, he said he believed he might make the same error a second time.

DR. G. H. MUNDT said there was a strong tendency to depend upon intranasal surgery in orbital abscesses secondary to nasal sinus infection. He was thoroly convinced that when Dr. Yerger limited himself to the combined procedure, that is external and intranasal, he might even do better if he limited his work to the external procedure. He questioned whether it was ever safe to work in the nose during acute sinus infections. In an occasional case of acute ethmoiditis with orbital abscess, before he knew enough not to do so, he had pulled off the middle turbinal. The cases had cleared up, but he thought it poor judgment, and he might have killed the patient.

DR. YERGER, in closing said he agreed with Dr. Brawley and also with Dr. Mundt. The X-ray was not infallible, and the more that was kept in mind, the better would be the results obtained. He performed an external operation; he saw no objection to removal of the middle turbinate intra-

nasally, preliminary to the external operation.

Posterior Chamber Cysts of Iris.

Dr. J. H. Roth, Kankakee, Ill., read the paper on this subject to be published.

Discussion. Dr. Robert Von der Heydt said that the case Dr. Roth referred to was like many others that came to him—he saw it only once. Since the slit lamp was available, it was not necessary to question whether a cyst was implanted or arose within the eyeball. It was always possible to find the wound of entrance in cases of implantation cysts.

Dr. Wm. C. Moncrieff asked whether these cysts could have arisen from the ciliary processes rather than from within the iris.

Dr. E. V. L. Brown said that if it were an implantation cyst, one should be able to follow ingrowing surface epithelium along a wound tract into the anterior chamber. For this, serial sections were indispensable. When eye tissues were turned over to a general pathologist to study, as was done in this case, such serial sections were seldom made, and very much lost.

Dr. Harry Gradle believed that many ophthalmologists did not know that the services of eye pathologists were at the disposal of anyone who wished to send a specimen to the Army Medical Museum at Washington. The specimen should be accompanied by a full history of the case, and it would be sectioned, studied by the best pathologists in the country and slides returned to the donor. This was being done without cost, for the purpose of enlarging the records and histories of the museum.

Dr. J. H. Roth, answering Dr. Moncrieff and closing the discussion, said that when one looked at the slide under the microscope, it would be seen that the origin of the cyst was from the very root of the iris. About the size of the eye—the vision was 15/40 corrected, so this could not have been a phthisis bulbi. Its going thru the general pathologist's hands, or because of the way it was fixed, whether ten per cent formalin or Zenker's, may account for the small size observed on the slide. However, it was of normal size and normal tension. Had he planned to have this eye sectioned, it would have been sent to an eye pathologist, and he was very sorry this was not done.

Clarence Loeb,
Recording Secretary.

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THE WILMER FOUNDATION OF JOHNS HOPKINS UNIVERSITY.

The justly famous eye hospitals and clinics of the old and the new world are crowded with helpless patients; for the first thing eye trouble does to its victim, is to destroy his earning power. So it is that Moorfields, the New York and Manhattan Eye Hospitals, those in Boston, Chicago, New Orleans and those of other large cities of this country, have all that they can do to keep their heads above water and make a precarious income meet the outgo, having little money; and no time to devote to scientific research.

But attention has been drawn to this situation; and in one of our most famous institutions of learning, the Johns Hopkins University of Baltimore, there has now been established a research institute for ophthalmology. By the friends of the hospital, and grateful patients of William Holland Wilmer of Washington, $3,000,000 has been raised toward this end.

Wilmer's very warm friends, grateful patients, and enthusiastic collaborators prosecuted the work of getting up an endowment fund with a great deal of ardor. Dr. Wilmer's modesty and reticence, even to his close personal friends who have begged for information for months on this subject, in order to publish this important news to the ophthalmic world, have delayed any complete anouncement of the facts.

The Johns Hopkins Univesity was desirous of developing its department of Ophthalmology; and when the trustees of the Wilmer foundation combined forces with the University, they achieved the desired end of raising more than $3,000,000. Over $1,500,000 of this amount was contributed by friends and patients of Dr. Wilmer; the other $1,500,000 was given by the general education board of the Rockefeller Foundation. Not only was the latter subscribed upon the contingency of an equal amount being contributed by personal contributions of American citizens, but most of the larger contributions were given on condition that Dr. Wilmer accept the headship of the new foundation. This will mean his giving up what is perhaps the most exclusive private practice in the world, and moving to Baltimore in order to direct the new institution at Johns Hopkins. He is also Professor of Ophthalmology in the medical department of Johns Hopkins and Ophthalmologist in Chief to Johns Hopkins Hospital.

At first, the former nurses' home will have to be converted into a temporary hospital; while the laboratory, etc., are being built. The scope of this work will embrace clinical ophthalmology, physiologic optics, immunology, serology, pathology and general research, including animal experimentation. After the laboratories are completed the Institute will function, not only as a hospital and out-door department; but instruction in ophthalmology will be given to undergraduates and graduates.

Many contributions, as aids to teaching and to the museum, have already been received by the foundation; and congratulations upon its proposed work have arrived from all parts of the world, with offers of assistance not only from ophthalmologists but from famous personages of this and foreign countries.

The esteem in which Dr. Wilmer is held makes this a living monument to his life work, in addition to the many high appointments which he has hitherto held with credit such as presidency and associate in many medical and scientific societies, and a leader of the ophthalmologists of the United States of America. He returned from the World War with the rank of Brigadier General, with the Distinguished Service Medal of America, Commander of the Legion of Honor of France, the confidence of Washington, of the United States Government and of the world. His coworkers in the science of Ophthalmology express to him their congratulations and best wishes for the carrying out of this noble work.

H. V. W.

THE HEIDELBERG CONGRESS.

In spite of the unfavorable financial conditions, the German Ophthalmological Society met again after a year with a large attendance at Heidelberg, the old meeting place, August 3rd, 4th and 5th. The evening of Sunday, August 2nd, members and guests met in a most informal manner in one of the dining rooms of Hotel Schiff. Old friendships were renewed and new acquaintances made while a meal or glass of beer was consumed. A more homelike gathering could hardly be imagined; a warm and hearty welcome was extended from all sides to the visiting Americans.

The scientific meetings were held on Monday, Tuesday and Wednesday, meetings beginning at 8:30 in the morning and at 3 in the afternoon. Forty-nine papers and twenty one demonstrations were on the program. Only two or three papers were omitted. Especially noteworthy was the great number of papers by the youngest members of the various universities. The character of the papers was of the highest, nearly all dealing with problems and general questions; most remarkable was the great absence of case reports. Discussion was very brief, because of the rule that case reports should be eliminated and that only questions of principle be discussed. The afternoon of Monday, August 3rd, was entirely devoted to the 21 demonstrations, all of which were finished in the allotted time, because of the thoro preparation of the speakers and the strict adherence to the subject. All papers and demonstrations were profusely illustrated by pictures thrown on the screen. The evenings of Monday and Tuesday were given to dinners at two places offering most beautiful views of Heidelberg and its surroundings. At each occasion very cordial relations were manifest between members and their guests. Sixteen American ophthalmologists, some with their families, were present at all functions. The president of the Society, Geheimrat Uhthoff, the secretary, Professor Wagenmann, Geheimrat Axenfeld, Hofrat Fuchs and others repeatedly expressed their welcome to the large number of visitors that had come from many countries: United States, China, Bulgaria, Greece, Switzerland, Austria, etc. The next meeting is to be held in 1927.

M. F.

THE SLIT LAMP AND BIOMICROSCOPY.

A few years ago there was introduced into ophthalmology a new instrument, the slit lamp, to be used in

conjunction with the binocular microscope. So much interest has been aroused that courses regarding it were given in several cities during the past year.

The busy doctor does not wish to add a new instrument, particularly a very expensive one, to those that have already been relegated to the store room. So he asks first, will the slit lamp add anything to his ability to make a diagnosis; and if so, will this occur sufficiently often to justify the time required for the routine use of the lamp. This is the question that those who have used this instrument for a considerable period should be prepared to answer.

More often the question is asked, what can be seen by this method that cannot be seen by any other. The answer is, not many things. But only a minor part of the value of the lamp is in seeing things that cannot be seen by other means. There are very few things that can be seen with a microscope with oil immersion, that cannot be distinguished with the usual high power magnification, but what pathologist would study bacteria without an oil immersion lens when that were obtainable! He sees better, things already visible with a lower power. So it is with the slit lamp. For example, very few of the products of inflammation visible with the slit lamp are not visible by other means, but with the former they are much better seen and their nature more easily differentiated.

As a routine part of the examination of each patient the use of the slit lamp is too time consuming to be justifiable, but one of the advantages of prolonged employment of the lamp is, that with this comes an ability to discriminate the cases in which the lamp may possibly be of service; and the busy practitioner will soon find himself confining its use to such cases. Calls for the lamp will occur almost as often as there will be calls for the perimeter. An experience of three years leads to the general conclusion that the instrument is not a necessity, but a very valuable adjunct to the equipment of the ophthalmologist.

L. T. P.

BOOK NOTICES.

Refraktion und Akkommodation des menschlichen Auges. Prof. Dr. A. Siegrist, Director of the University Eye Clinic, Bern. Cloth, large 8 vo., 154 pages, 108 illustrations, many in colors. Berlin, Julius Springer, 1925.

The number of different aspects of refraction and accommodation that different authors emphasize, illustrates the greatness and many sidedness of the subject of refraction. In this particular monograph, the author has avoided any use of algebraic formulas, presenting the mathematic side by geometric diagrams; and he has especially dwelt on the important practical subjects of glasses and visual acuity. His appreciation of relative importance, however, leans toward the pathologic anatomy of the lesions associated with myopia, a rather common tendency in the works on this subject that have appeared in the German language. Of the 20 colored pictures of the fundus, 19 represent the pathologic appearances seen in the myopic eye, and several other colored plates represent the histologic changes that belong with myopia.

The book begins with an introduction of 10 pages, with 6 illustrations, 4 in colors, devoted to the anatomy of the normal human eye and the normal eyeground. This is one of the most brief, clear, satisfactory presentations of the essential facts, that we have encountered in any language. Then come 8 pages on dioptrics, with 4 illustrations that include 17 different diagrams showing the relations of foci to the refracting surfaces and media that produce them. In 16 of these the course of the rays of light is emphasized by representation in red lines. After this glasses are discussed in 28 pages; with 34 illustrations as their numbers indicate. But 3 of these, each occupy a page, and together they represent 42 different forms and details of spectacles and eyeglasses.

The section on accommodation occupies 14 pages, having 6 of these multiple figures. Fig. 46, called sight with diffusion circles, includes 9 separate illustrative diagrams. It is succeeded by the section on visual

acuity, 14 pages with 8 illustrations. This completes the first half of the book.

The remaining 74 pages, with their 47 illustrations, are devoted to the optical or refractive defects of the eye. Of this, 8 pages are given to hyperopia, 18 to astigmatism, and 47 to myopia with its pathologic conditions revealed by the ophthalmoscope and its pathogenesis and prophylaxis. There is a fair table of contents, but no index. To get command of its contents, one must read the book thru and then remember its arrangement. References to the literature are supplied in foot notes, particularly in the portion relating to myopia.

To ophthalmologists who read German, this book is well worth having. Teachers of ophthalmology, even tho they do not read German, will find it especially suggestive as to methods of presenting by diagrams, points in refraction that are often found difficult to render clearly intelligible to the student. They can scarcely afford to be without it. Its price, 18.60 gold marks, might be well expended for the 20 colored plates of the fundus alone; for these are among the best that have been published since the world war, altho they are printed with the text and not on separate plate paper.

E. J.

Ocular Therapeutics. Dr. Ernst Franke, Prof. of Ophthalmology at the University of Hamburg. Translated by **Clarence Loeb,** A. M., M. D., Oculist to the Michael Reese Hospital, Chicago, Ill. Cloth, octavo, 183 pages. St. Louis, the C. V. Mosby Co. 1925.

In these days of crowded reading, to remember all one would like to remember, of what he has read, is a rare accomplishment. To be able to summon up for each emergency all that has passed thru his mind as worth while regarding therapeutics, even in a limited field like ocular therapeutics, is impossible to most who have read widely even for a few years. It becomes necessary either to form one's own list or collection of the articles he would like to refer to, or to utilize the lists and collections of therapeutic facts made by some one else, to keep at command what one has already found for himself in the literature. Such a collection, well indexed and up-to-date, is presented to English reading oculists in this volume.

It is something more than a condensed epitome of what a reader might know if he could remember all he had read about ocular therapeutics. As the author says in his preface, it is "an attempt to give in a few words our present knowledge of the treatment of diseases of the eye." As the translator says in his preface "there are available to the Engligh speaking oculist very few books to which he can go for information as to the treatment of his patients, which, after all, is the point which concerns the latter most."

After a 4 page introduction, Chapter I, which emphasizes the pathologic unity of the body, the book is divided into two parts, a General part and a Special part. In the former, Chapter II (68 pages) takes up general treatment, under the headings; tuberculosis, syphilis, serotherapy and organotherapy, paraspecific protein body, irritative therapy, ray and light therapy, electric treatment, medical treatment, general use of cold and heat, baths, bath and spring cures. Chapter III deals with local treatment, under the headings; mechanical treatment, warmth and cold, light and ray treatment, local medical treatment, serotherapy and electrotherapy.

In Part II, Chapter IV (22 pages), the different pathologic conditions that may require treatment are given under the headings (15) of the parts involved; as, lids, conjunctiva, lacrimal apparatus, etc. Under each of these are grouped the particular conditions to be treated, as under optic nerve; neuritis, choked disc, retrobulbar neuritis, atrophy, amblyopia and amaurosis, and tumors. The book concludes with an 11 page, double column index, with something like 220 references to a page, which makes this a real book of ready reference.

Franke's book has been before its German readers for over a year; so that, like textbooks in general, it cannot be depended upon for the very latest additions to our therapeutic re-

sources, especially some of American origin and popularity, as insulin and mercurochrome. On the other hand the many drugs of German origin and exploitation are well presented. Thus afenil, chlorylen, caseosan, scarlet red, etc. Opening the second chapter is a most excellent resumé of tuberculin treatment and the various preparations of tuberculin used, 8 in number. A little farther on we come to a similar account of the newer mercurial and arsenic preparations, for the treatment of syphilis. In both sections are given the most valuable practical suggestions as to administration.

For the recent graduate in medicine this book will help to put him most quickly in command of the therapeutic resources of this special branch of practice. To the busy, experienced practitioner it offers suggestions of new remedies of which he has felt the need. Our therapeutic resources cannot be too extensive, their mobilization for instant use cannot be too complete. In this direction we always need all the help we can get, and here are offered many suggestions, a single one of which in a single emergency may be well worth the price of the book, and many times the slight effort of consulting it. E. J.

The Ophthalmic Year Book. Volume 21, edited by **William H. Crisp,** assisted by 33 collaborators. Cloth, 334 pages, 10 illustrations. Chicago, The Ophthalmic Publishing Company. August, 1925.

Bearing in mind the daily pressure of reading matter upon time, attention and memory, the editing which reduces its amount and increases its value should be highly prized. When we remember that 20,000 or 30,000 pages are looked over for the preparation of this volume, judgment in selection and brevity of statement seem the highest possible virtues for such a publication. The smaller size of 1925 Year Book as compared with some of its predecessors ought to be counted a superiority.

Hippocrates, 150 years after the introduction of writing into Greece, said, "Art is long, life is short." What should we think of our situation, when the issue from the printing presses in a single day exceeds that of the first 150 years following its invention. Not the number of printed pages that can be turned out, but the slowly evolving capacities of the human mind limit the total of profitable reading it is possible for one to do. Should not the publications that furnish the most of information and suggestion in the fewest pages, making the smallest demand on our time and effort, be given the highest place in our estimates of books?

This volume is 5/6 the thickness of the Ophthalmic Year Book of 1925. It has 99 fewer pages. Its bibliographies contain references to 15 per cent fewer papers. But it represents the study of an equally great literature, more hours spent upon its preparation, better arrangement and coordination of the matter it contains. Most papers not mentioned in the Year Book contain no material worthy of perpetuation in its pages and their titles are already given in the lists of "Current Literature." For these characteristics this volume is superior to all its predecessors, it has made an advance in brevity, one of the ideals of all literary expression. The value of this condensation depends of course on the judgment of the collaborator or editor, as to what shall be included and what shall be omitted. But it also depends on the use that the reader would make of the Year Book. If he wishes to make quotations from it to incorporate in a paper, to be read before a meeting, or published, there may be no advantage in having the main thought isolated from its related matter. If he is a student who wants to learn everything possible about a particular subject, it might be an advantage to have the original paper quoted at length, rather than have its essential ideas reproduced in brief statement and stripped of what is less important.

But if the reader only wishes the important experience and suggestions of a writer, to apply to the needs of his own practice, the separation of these from what he does not need is a real service. The bulk of subscribers for the Year Book belong to this last class. They should be served first by

the publication; the writer, or special student, can use the volume as a guide to the kind of material he wants; and then he will get more help by going to original sources than he could from a more bulky Year Book, that ignored the special needs of the larger class of its readers.

Three changes introduced in the present volume deserve notice. Thruout the digests of the literature figures are placed, that help the reader to go from the bibliography to the exact reference to each paper in the digest. This is an added convenience. The bibliography is still arranged alphabetically by authors' names, so that it is easy to find all that a certain author has published on a certain subject, or related subjects, in the year; and the number given in the digest indicates exactly which of his papers the statement is taken from.

In the index of authors' names the references given are to the *chapters* in which papers published by each writer are noticed; not, as heretofore, the *pages*. Turning to the bibliography at the head of the chapter the title of each paper is found under the author's name in alphabetic order; and the number given each paper indicates where in the digest it is cited. This furnishes a very complete and direct way of finding what you are after, making this a better reference book than we have had before. Each chapter now is given a number, thus: Chap. I, General Methods of Diagnosis, Chap. XV, the Retina. These chapter numbers, placed at the head of each left hand page, make it always easy to find the particular chapter desired.

A list of books relating to ophthalmology, that have appeared during the last year (1924), is added to Chapter XXXII, on Education, History and Institutions. It will be very helpful; giving as it does, exact titles, edition, publisher, size of book, and how illustrated, with a reference to the journal and page where the book has been noticed. With this help every reader can make an intelligent application of whatever money he can spend, from year to year, on new books relating to ophthalmology. Let us repeat; this year Year Book is better than any of its predecessors.

E. J.

Estudios de Oftalmologia. Dr. Jose de Jesus Gonzalez. Corresponding member of the National Academy of Medicine of Mexico, the Mexican Ophthalmological Society, etc. Jubilee Book, volume 1, paper, small quarto, 550 pages, illustrations, including 3 colored plates, Mexico, Franco-Mexican Press.

This work is the first volume of a series of reprinted ophthalmologic papers by Dr. Gonzalez. It is published to celebrate the 25th anniversary of his doctorate. The titles of the various papers are: Hygiene of Light and Prophylaxis against Blindness (19 p.); Prophylaxis and Treatment of Purulent Ophthalmia of the New Born (17 p.); Purulent Ophthalmia of the New Born and the Responsibility of the Professors of Obstetrics (7 p.); Measures Proposed for the Diminution of the Frequency of Blindness in our Country (38 p.); The Therapeutic Value of Instillations of Quinin in Suppurative Affections of the Anterior Segment of the Eye (7 p.); Therapeutic Notes (12 p.); Two New Drugs in Ocular Therapeutics (16 p.); Treatment of Neuritis Optica (38 p.); Electricity as a Hypotonic Agent in Ophthalmology (3 p.); Physiotherapy in Ophthalmology (24 p.); Autohemotherapy in Spontaneous Juvenile Vitreous Hemorrhages (8 p.); Action of Tuberculin on Healthy and Diseased Eyes (41 p.); Parenteral Injections of Milk in Ocular Therapeutics (57 p.); Notes on a Method of Ablation of Total Staphyloma of the Cornea (5 p); Some Ocular Complications of Influenza (8 p.); Monocular Ophthalmoplegia Interna During a Puerperal Infection (4 p.); Ocular Complications of Mexican Typhus or Tabardillo (26 p.); Ocular Symptoms Appearing During a Febrile Affection of a Recurrent Type (7 p.); Ocular Complications Appearing During Convalescence from Chronic Fevers of the Paratyphoid Type (7p.); Ocular Symptoms of Poisoning by the Sting of Scorpions (17 p.); Frequency of Hereditary Ocu-

lar Syphilis and a Study of Some Rudimentary Stigmata (29 p.); Keratoglobus Following a Tertiary Dystrophy of the Corneal Limbus (5 p.); Influence of Trauma on the Appearance of Interstital Keratitis (4 p.); Leproma of the Iris, Cured by Radiotherapy (9 p.); Iconography of Ocular Leprosy (25 p.); Relation Between Ocular Pathology and the Internal Secretions, (2 p.); The Hepatic Hormones and Epidemic Idiopathic Hemeralopia (15 p.); Endocrin Disturbances in the Etiology of Cataract (34 p.); Acute Suprarenal Insufficiency and Avitaminosis in the Pathogenesis of Keratomalacia (17 p.). Each paper is followed by its bibliography, and there is an index of the authors quoted, as well as an index to the papers appearing in this volume.

In reviewing this book, one regrets that so few American oculists read Spanish. There is a tremendous amount of valuable ophthalmic information which is inaccessible to the great majority of us. Interesting cases, histories, valuable statistics and important conclusions are detailed, but unfortunately can be utilized at first hand by few except our Hispano-American and Spanish colleagues. Considering the large number of these and the amount of valuable medical literature coming from them each year, a working knowledge of Spanish might well be one of the requirements for a medical degree.

The papers included in this volume have all appeared at various times in medical journals and transactions, and will not be reviewed here. Attention should be called to the paper on "Measures Proposed for the Diminution of the Frequency of Blindness in Our Country." The papers dealing with leprosy are well illustrated and interesting, tho hardly of importance to American oculists. The grouping of the papers into the various divisions—Hygiene of Light and Prophylaxis of Blindness, Ocular Therapeutics, Ocular Surgery, Ocular Leprosy, Endocrin Disturbances and Ocular Pathology, and the various others for succeeding volumes, add greatly to the value of the book. C. L.

This volume commemorates the twenty-fifth anniversary of the granting of the medical degree to Dr. Gonzalez in 1897. It includes monographs and papers published by him since 1909 in various transactions and journals, and is presented by Daniel M. Velez, M. D., Director of the Anales de la Sociedad Mexicana de Oftalmologia Y Oto-Rino-Laringologia. Perhaps no more appropriate, interesting and useful celebration of the career of this able and prolific writer on ophthalmology could be desired. His work has been done at Leon in the province of Guanajuato, a city of 70,000 inhabitants; which he is making as well known to ophthalmic readers, as the capital city of his country. He needs no introduction to the regular readers of this journal. But his activity in ophthalmic science and practice deserves recognition, on the part of his English speaking colleagues.

The volume opens with a prologue of pages, devoted to professional development; with an appreciative account of the ophthalmologists of Mexico and the scientific work they have done in the last 30 years, individually and thru their organizations and institutions. From this prologue we learn that this volume is only a part of the projected work, subjects to occupy one or two more volumes being mentioned.

The different articles thus brought together vary widely in character and nature of material thus published. That on the hygiene of vision is almost a systematic treatise. Under ocular surgery the single brief contribution is a report of a case of ablation of corneal staphyloma. Under ocular therapeutics, there are 11 different notes on the employment of different remedies for various clinical conditions. In some of the papers there is a fairly complete review of the recent literature bearing on the subject. Others merely record personal observations. The three colored plates belong to the article on ocular leprosy. The excellence of the typography, paper and press work is quite up to the standard for a commemorative volume. Its readers will look to Mexico with increased expectancy for volumes on ophthalmology quite worth having. E. J.

Directorio Medico Mexicana. Published under the direction of Dr.

Daniel M. Velez. Director of the Mexican Ophthalmologic and Otorhino-laryngologic Society. Paper, small quarto, 676 pages. Mexico, D. F., Imprenta Victoria, S. A.

This is something more than a medical directory such as is known in English speaking countries. It is arranged in some 20 sections. The first describes the 5 medical schools of Jalisco, Nuevo Leon, Michoacan, Pueblo and Mexico, the National Dental School and the National School of Veterinary Medicine of Mexico. Section II describes the hospitals, III the Sanitoria, IV the Medical associations and allied scientific societies. From the account of the Mexican Ophthalmologic and Oto-Rhino-Laryngologic Society we learn that it was organized in 1893. It has held monthly and annual meetings, and has to its credit various publications thru the Anales de Oftalmologia edited by M. Uribe Troncoso and Daniel M. Velez.

Other sections deal with laboratories, health department and regulations, legal regulations regarding Medical practice, a bibliography of Mexican medical works, including 11 on ophthalmology published from 1908 to 1923, the professional directory including physicians, general and special, dentists, pharmaceutists, veterinarians, etc., (122 pages). There is an alphabetic list of medicinal preparations, and two sections devoted to commercial interests.

In some of the sections, there is a good deal of historic interest, and there are many illustrations, chiefly reproductions of photographs of buildings, faculties, hospital staffs, famous paintings, as of Pasteur's first inoculation of a human being for rabies, and a few of prominent Mexican physicians. It is a volume that fully justifies its existence: and should find a place in all public medical libraries. E. J.

CORRESPONDENCE.

FIRST GRADUATE COURSE IN OPHTHALMOLOGY GIVEN IN CHINESE LANGUAGE

The first postgraduate course in ophthalmology to be given in the Chinese language took place at the Peking Union Medical College, Peking, China during the period January 26 to February 21, 1925. This event is epochal in the history of medicine in China. The course was conducted in the northern Mandarin dialect altho none of the six instructors is a native of North China.

The course was carried on under the direction of Associate-Professor T. M. Li, who was assisted by the junior members of the staff consisting of Dr. W. P. Ling, Dr. T. P. Lee, and Dr. T. Y. Lai. Dr. T. C. Pa, a former member of the staff and at present Assistant-Professor and Head of the Department of Ophthalmology in the Shantung Christian University Medical School, Tsinan, Shantung, gave valuable assistance during the whole of the course. Dr. Howard, who returned from a medical conference at Hongkong shortly before the end of the course, participated by giving two illustrated lectures on the bacteriology of the eye. The intention of the instructors was to make the course of the greatest practical value to the students. The daily program covered seven hours of work, and besides a large number of clinical and surgical demonstrations, included refraction, ophthalmoscopy, perimetry, operative surgery on pigs' eyes, eye pathology and bacteriology.

Heretofore all the undergraduate and postgraduate teaching at the Peking Union Medical College has been carried on thru the medium of the English language. But there has been such an insistent demand for postgraduate instruction on the part of physicians who received their medical training in the Chinese language and who understand English but little or not at all, that the Director of the College finally sanctioned a course in ophthalmology largely as an experiment. There were far more applications than could be accepted. The following nineteen physicians were admitted and took the course:

Dr. Yang C. Chang
Dr. Yu C. Chang
Dr. H. L. Chen
Dr. K. Y. Chen
Dr. C. Y. Chu
Dr. S. Y. Han
Dr. L. H. Hsu
Dr. C. H. Hu
Dr. P. S. Lee
Dr. C. C. Li
Dr. K. H. Li
Dr. C. L. Liu
Dr. Y. S. Liu
Dr. C. S. Shih
Dr. C. Y. Sung
Dr. T. K. Tsang
Dr. C. T. Wang
Dr. Cora Wang
Dr. F. H. Yao

Nine of the eighteen provinces in China were represented by this group, viz: Chihli 8, Fukien 1, Hupeh 1, Kiangsi 1, Kiangsu 1, Kwangrung 2, Manchuria 2, Shantung 2, and Szechuan 1.

The course proved such a success as shown by the results of an examination as well as by the unabated interest on the part of the students thruout the whole course, that it is the plan henceforth to alternate the customary Chinese New Year's course by giving the course one year in the English language and the next year in the Chinese Mandarin.

HARVEY J. HOWARD.

Peking Union Medical College.

NEWS ITEMS

DEATHS.

Dr. George H. Savage, Memphis, Tennessee; aged forty-nine, died July thirtieth.

Dr. Walter B. Johnson, Paterson, New Jersey, aged sixty-nine, died, suddenly July thirtieth of angina pectoris.

Dr. Adolph Vossius, emeritus professor of ophthalmology, Giessen; aged seventy-one, died recently..

Dr. Claude M. Pearce, Portland, Oregon; aged forty-eight, died June twenty-fourth, of interstitial nephritis.

SOCIETIES.

The Twin Lakes District Medical Society of Iowa held a diagnostic clinic July sixteenth at North Twin Lake, Rockwell City. Among the speakers was Dr. Harold Gifford of Omaha.

At the annual meeting of the Idaho Medical Association held at Pocatello, September third to fifth, Dr. Edward Jackson of Denver delivered an address on "Examination and Management of Recent Injuries to the Eye."

At the recent annual meeting of the Houston Ophthalmological and Oto-Laryngological Society, Dr. Palmer M. Archer, Houston, Texas, was elected president; Dr. Nicholas L. Dudley, Goose Creek, vice-president; and Dr. W. Marcel Strozier, Houston, secretary.

At the annual meeting of the Buffalo Ophthalmological Club, Dr. L. M. Francis was elected president, Dr. Harry M. Weed, vice-president and Dr. A. F. Luke, secretary. During the past year, Dr. D. N. Dennis of Erie, Dr. Conrad Berens of New York, Dr. Allen Greenwood of Boston, and Dr. Luther Peter of Philadelphia, were honor guests.

A Slit Lamp Society has been organized by a group of nine Pittsburgh ophthalmologists. Temporary officers chosen were Dr. W. W. Blair, chairman; Dr. George H. Shuman secretary. Professor Leonard Koeppe, of the University of Halle, Germany, was elected an honorary member.

The Colorado Congress of Ophthalmology and Oto-Laryngology with the Summer Graduate Course, covering a period of two weeks, was held in Denver in August. Fifty-five students were enrolled in the graduate course and 113 registered at the Congress. There was a very general feeling that Denver should continue to give this instruction.

At the meeting of the Pacific Coast Oto-Ophthalmological Society at Vancouver, the following officers were chosen: President, Dr. Kasper Pischel; First Vice-president, Dr. Glen Campbell; Second Vice-president, Dr. Edward Neher; Secretary-Treasurer, Dr. Walter F. Hoffman.

AMERICAN BOARD OF OTOLARYNGOLOGY

The next examination given by the American Board of Otolaryngology will be held at the Cook County Hospital, Chicago, on October 19th, 1925. Application should be made to the Secretary, Dr. H. W. Loeb, 1402 South Grand Boulevard, St. Louis, Missouri.

PERSONAL

Dr. Victor C. Smith has been appointed ophthalmologist to the new Southern Baptist Hospital of New Orleans.

Dr. Edward V. L. Brown has been appointed professor and head of the department of ophthalmology of the medical department, University of Illinois.

Dr. F. J. Pinkerton recently returned to Honolulu after spending several months doing postgraduate work in the large cities on the mainland.

Dr. Kasper Pischel of San Francisco was recently elected president of the Pacific Coast Oto-Ophthalmological Society and San Francisco chosen as the place of the 1926 meeting.

Dr. John O. McReynolds, Dallas, Texas, spent several weeks' vacation in the Hawaiian Islands. He thinks the islands an ideal place for a tired professional man to take a rest.

Dr. Edgar W. Alexander, San Francisco, is spending several months in the Hawaiian Islands and expresses himself as being much pleased with the surf riding and other sports.

Dr. Wm. C. Finnoff of Denver and Dr. E. Bribach of Atchison, Kansas, have returned from a trip abroad, where they spent a short time in France, Holland, Belgium and Germany and attended the Congress of Ophthalmology in London.

Dr. L. M. Gurley of Johnstown, Pennsylvania, has just returned from Europe, after spending two months visiting the main eye clinics. He visited Berlin, Brussels, Strasbourg, Paris, London, Manchester, Dublin, Belfast, Glasgow, and Edinborough.

Dr. Wade E. Carson of Pittsburgh, Pennsylvania, sailed from New York on July second to attend the Ophthalmological Congress in London and the other opthalmologic meetings abroad this summer.

Dr. Edward B. Heckel was elected president of the Pittsburgh Ophthalmological Society for the fourteenth successive year. Other officers reelected for the ensuing year were Dr. G. E. Curry, vice-president and treasurer; Dr. George H. Shuman, secretary.

Dr. John E. Weeks of New York, delivered a series of lectures at the Summer Graduate Course of the Colorado Congress on Surgical Technique of Operations; Dr. Francis Lane of Chicago gave three lectures on Pathological Anatomy of Deep Scleritis, Sympathetic Inflammation and Atrophy of Eyeball.

At the annual meeting of the British Medical Association at Bath. July 21st, the Middlemore Prize was presented to Basil Graves for the contribution which he has made to the knowledge of ophthalmologists of microscopy of the living eye, especially in relation to the use of the slit lamp.

Dr. William H. Wilmer, director of the new Wilmer Institute, visited Johns Hopkins Hospital, Baltimore, August fifth, to inspect changes in construction which are being made in that portion of the institution devoted to the institute which will open October first. Appointments are now being made. Dr. Cecil H. Bagley will act as first assistant to Dr. Wilmer and will be the first resident physician. Dr. Jonas Friedenwald will be the pathologist and will conduct the research work in eye diseases.

Dr. M. Uribe Troncoso has returned to New York City, after a two months vacation in Cuba and Mexico. While in Mexico City he was invited by the National University to give a course on Internal Diseases of the Eye. This course lasted twelve days and was held in the Ophthalmic Hospital of La Luz. He was entertained at a dinner by the Mexican Ophthalmological Society where he gave a lecture on "Gonioscopy."

Professor Leonard Koeppe gave a course in Slit Lamp Microscopy of the Eye to a group of Pittsburgh, Pennsylvania, ophthalmologists in June. The course was given in the Eye Clinic of the University of Pittsburgh. Professor Koeppe has been invited to return to Pittsburgh this coming winter to give a more advanced course to the same group. He also gave a number of courses in Slit Lamp Microscopy in the Knapp Memorial Hospital and the Manhattan Eye and Ear Hospital, New York City, with a large number of students, some of them well known oculists of the metropolis. Professor Koeppe has given courses in Cincinnati, St. Louis, and Cleveland and will return to New York for another series of lectures.

MISCELLANEOUS.

The Harlem Eye, Ear, and Throat Hospital, under the will of the late Amelia Brady, was bequeathed $1,000.

Under the will of the late Mrs. Martha G. Hervey, the New York Association for the Blind was bequeathed $2,500.

The Maine Institution for the Blind received $500 by the will of the late Miss Eliza Jordan of New Gloucester, Massachusetts.

The entire residuary estate of Dr. Nichols R. Dann, estimated now at about $50,000 will go, on the death of his widow and sister-in-law, to the New York Eye and Ear Infirmary.

As a result of the survey made by experts from the United State Public Health Service into the question of trachoma in the state of Indiana, the State Board of Health is prepared to say that trachoma is not prevalent in the schools of Indiana or among the general public.

The Chicago Lighthouse conduct a workshop for the blind at 3323 West 22nd Street. Physicians are requested to report to that institution blind people who need its help. They will receive training without expense in typing, piano tuning, dictaphone operating, mechanical assembling, folding, packing, sorting, stacking and wrapping of articles, weaving on hand looms, and other work Patients should be referred just before impending blindness or soon after. Later they lose courage and it is difficult to regain their selfconfidence.

In a study of eye hazards in industrial occupations, the National Committee for the Prevention of Blindness recently concluded that, of the 100,000 blind persons in the United States, about 15,000 have lost their sight in the pursuit of industrial occupations, and there is, in addition, a much larger number of persons whose vision has been so impaired in the industries that they will be handicapped thru life. The metal manufacturing industries are the source of the greatest number of serious eye injuries. In Pennsylvania, the coal mining industry ranks first as a cause of industrial blindness; in Wisconsin, "hand tools" is the greatest single cause, and in a large shipbuilding company 38 per cent of all cases treated in its dispensary were eye injuries. The full report of this committee is a volume of 250 pages and may be secured from the Committee for the Prevention of Blindness, 130 East Twenty-second Street, New York. (A. M. A. Journal.)

Current Literature

These are the titles of papers bearing on ophthalmology. They are given in English, some modified to indicate more clearly their subjects. They are grouped under appropriate heads, and in each group arranged alphabetically, usually by the author's name in heavy-faced type. The abbreviations mean: (Ill.) illustrated; (Pl.) plates; (Col. Pl.) colored plates. Abst. shows it is in an abstract of the original article. (Bibl.) mean bibliography and (Dis.) discussion published with a paper.

BOOKS.

Ballantyne, A. J. A pocket book of ophthalmology. 2nd Edition, 1924. Edinburgh: E. and S. Livingstone. 8vo., p. 144. Med. Jour. of Australia, July 4, 1925, p. 14.

Jackson, E. and Crisp, W. H. Ophthalmic Year Book, Vol. XX. 9 ills. Pub. by the Ophthalmic Publishing Co., Chicago, Sept., 1924. A. J. O., 1925, v. 8, p. 670.

Ryer, E. L. Ophthalmology. Pub. by the Optical Publishing Co., New York City, 1925, 220 p., 59 ills. A. J. O., 1925, v. 8, p. 669.

Transactions of the Pacific Coast Oto-Ophthalmological Society, 12th Annual Meeting, Portland, Ore., 1924. A. J. O., 1925, v. 8, pp. 669-670.

Transactions of the Section on Ophthalmology of the American Medical Association. 75th Annual Session, Chicago, Ill., 1924. A. J. O., 1925, v. 8, p. 670.

DIAGNOSIS.

Bayley, W. D. Hysterical amaurosis. Hahnemann Month., 1925, v. 60, pp. 479-483.

Collins, S. D. and Britten, R. H. Variation in eyesight at different ages as determined by the Snellen test. Pub. Health Rep., Dec., 1924, p. 3189-3194.

Dancy, A. B. Ophthalmoscopic examination. Jour. Tenn. State Med. Assn., 1925, v. 18, pp. 13-17.

Ferree, C. E., Rand, G., and Wentworth, H. A. Blind spot for achromatic and chromatic stimuli. (3 tables, 5 ills., bibl.) A. J. O., 1925, v. 8, p. 620-625.

Fortin, E. P. Seeing the movements of blood corpuscles in one's own eye. Semana Med., 1925, v. 32, pp. 126-129.

Koby, F. E. Modification of slitlamp. (1 ill.) Rev. Gén. d'Opht., 1925, v. 39, pp. 53-59.

Ribas Valero, R. Visual acuity. Anales de Oft., 1925, May, pp. 275-294.

Vogt. Ophthalmoscopy with red free light. Münch. med. Woch., 1925, v. 72, pp. 1101-1103.

Wasielewski. Photography of eyes of living animals. Klin. M. f. Augenh., 1925, v. 74, p. 777.

Wendt, W. A new tonometer. (2 ills.) Klin. M. f. Augenh., 1925, v. 74, pp. 604-612.

THERAPEUTICS.

Corbett, J. J. Radium therapy in nonmalignant eye conditions. Med. Rev. of Rev., 1925, v. 31, pp. 327-342.

Gilbert. Ultraviolet treatment of ocular disease. Klin. M. f. Augenh., 1925, v. 74, p. 781.

Lemay, P. and Jaloustre, L. Comparative action of atropin and the total alkaloids of belladonna. Lancet, Aug. 1, 1925, p. 241.

Lemoine, A. N. Antipneumococcic properties of ethylhydrocuprein hydrochlorid (optochin) in vivo. (Bibl.) A. J. O., 1925, v. 8, pp. 609-612.

Lundsgaard, K. K. K. Sanocrysin in ophthalmology. Dansk. Ophth. Selskabs Forhand., 1924, p. 32.

Mayer, E. G. General roentgenology in ocular therapeutics. Klin. M. f. Augenh., 1925, v. 74, pp. 612-622.

Metzger, E. Sodium iodid in ophthalmology. Klin. Woch., 1925, v. 4, p. 1164. Abst., J. A. M. A., 1925, v. 85, p. 554.

Moron, J. Bismuth in ocular therapeutics. Arch. de Oft. Hisp.-Amer., 1925, v. 25, pp. 443-450.

Seggern. Corneal changes from nitrat of silver. Klin. M. f. Augenh., 1925, v. 74, p. 778.

OPERATIONS.

Bell, G. H. Cataract eye speculum. (1 ill.) J. A. M. A., 1925, v. 85, p. 268.

PHYSIOLOGIC OPTICS.

Bozzoli, A. Size of macula and extent of direct vision. (4 ills., bibl.) Ann. di Ottal. e Clin. Ocul., 1925, v. 53, p. 794-807.

Evolution of human vision. Lancet, July 25, 1925, pp. 184-185.

Fougerat. Man and light. Paris Thesis, 1925.

Hartline, H. K. Electrical response to illumination of eye in intact animals, etc. Amer. Jour. Phys., 1925, v. 73, pp. 600-613.

Hofmann, F. B. Space perception of eyes. (155 ills.) 667 p. Berlin. Julius Springer, 1920.

Parsons, J. H. Foundations of vision (Bowman Lecture). (Bibl.) Lancet, July 18, 1925, pp. 123-130.

REFRACTION.

Adler, F. H. Crampton accommodation rod. (3 ills.) A. J. O., 1925, v. 8, p. 634.

Bohnenberger. Double refraction (irregular astigmatism). Klin. M. f. Augenh., 1925, v. 74, p. 770.

Camchansky. Transient myopia. Paris Thesis, July, 1925. Abst., Gaz. des Hôp., 1925, v. 98, p. 888.

Erggelet. Accommodation. Klin. M. f. Augenh., 1925, v. 74, p. 771.

Eye-strain. B. J. O., 1925, v. 9, pp. 409-410.

Henker. Instrument for testing exactness of spectacles. Klin. M. f. Augenh., 1925, v. 74, p. 770.

Kapuscinski, W. Synergy of mechanism of accommodation of the two eyes. (Bibl.) Rev. Gén. d'Opht., 1925, v. 39, pp. 104-107.

Nakajima, C. Influence of stenopaic slit on central vision. Rev. Gén. d'Opht., 1925, v. 39, p. 19.

Plange, O. Jr. Spherical refraction in growing rabbits. (4 ills.) Klin. M. f. Augenh., 1925, v. 74, pp. 700-710.

Robinson, J. Traumatic myopia. A. J. O., 1925, v. 8, pp. 654-655.

OCULAR MOVEMENTS.

Adrogue, E. Disturbances of external ocular motility. Semana Med., 1925, v. 32, pp. 1369-1398.

Dupuy, J. Associated paralysis of cranial nerves. Paris Thesis, 1925.

Ficher, E. M. Binocular triplopia after operation for strabismus. Russian Ophth. Jour., Jan., 1925. Rev. Gén. d'Opht., 1925, v. 39, p. 183.

Leri, A. Oculomotor paralysis with facial hemiatrophy. Soc. d'Opht. de Paris, 1925, June, p. 259-264.

Meyer, S. J. Traumatic paralysis of depressors of both eyes. (Dis.) A. J. O., 1925, v. 8, pp. 661-662.

Miners' nystagmus. Lancet, Aug. 1, 1925, p. 260.

Ohm, J. Demonstration of nystagmus, etc. (Dis.) Zeit. f. Augenh., 1925, v. 56, pp. 64-67.

Orlandini, O. Nystagmus with disturbance of associated eye movements. Lettura Oft., Sept., 1924, pp. 441-456.

Disturbance of ocular movements in brain disease. Lettura Oft., 1924, Nov., pp. 539-551.

Post, L. Unilateral nystagmus. (Bibl.) A. J. O., 1925, v. 8, pp. 632-633.

Roussy, Bollack and Levy. Monocular nystagmus with myoclonus. Soc. d'Opht. de Paris, 1925, May, p. 228.

Stanka. Voluntary nystagmus. Klin. M. f. Augenh., 1925, v. 74, p. 787.

Thomas, A. Rotary nystagmus. Soc. de Neurol., June 4, 1925. Abst., Gaz. des Hôp., 1925, v. 98, p. 904.

Tinel. Intermittent paralysis of the third nerve associated with facial neuralgia. Soc. de Neurol., June 4, 1925. Abst., Gaz. des Hôp., 1925, v. 98, p. 903.

Young, H. B. Alternating squint. J. A. M. A., 1925, v. 85, p. 378.

Zabaleta, H. O. Modification of Maddox treatment of strabismus. Arch. de Oft. Hisp.-Amer., 1925, v. 25, pp. 432-443.

CONJUNCTIVA.

Aubaret. Surgical treatment of granulations in trachoma. Rev. du Trachome, 1925, v. 2, Jan., pp. 6-15.

Bonfiglio, V. Surgical treatment of trachoma. Lettura Oft., 1924, Oct., pp. 502-503.

Canitano, S. Diphtheritic and pseudomembranous conjunctivitis. Lettura Oft., 1924, Oct., pp. 489-502.

Castro de la Jara, R. and Castresana, A. Treatment of trachoma with radium. Arch. de Oft. Hisp.-Amer., 1925, v. 35, pp. 417-432.

Clapp, C. A. Pemphigus. A. J. O., 1925, v. 8, p. 649.

Cozzoli, G. Trachoma and conservation of vision in schools. Rome, 1925.

D'Alessandro, A. Trachoma. Semana Med., 1925, v. 32, pp. 109-122.

Demaria, E. B. Granular conjunctivitis in Argentine Republic. Rev. du Trachome, 1925, v. 2, Jan., pp. 27-55.

Engelking, E. Swimming bath conjunctivitis, trachoma, blennorrhea and gonorrhea. (3 ills.) Klin. M. f. Augenh., 1925, v. 74, pp. 622-634.

Fallows, H. D. Primary unilateral diphtheritic conjunctivitis. (1 ill.) A. J. O., 1925, v. 8, pp. 635-637.

Falta, M. Abrasion of conjunctiva in treatment of trachoma. Arch. f. Augenh., 1925, v. 96, pp. 432-436.

Garcia Mansilla, S. Parinaud's conjunctivitis. (3 ills.) Anales de Oft., 1925, v. 1, Jan., pp. 27-33.

Guibert. Acute and chronic ocular pemphigus. Clin. Opht., 1925, v. 29, pp. 382-386.

Hoffmann, W. Histology of vernal conjunctivitis. (2 ills.) Zeit. f. Augenh., 1925, v. 56, pp. 21-34.

Kubik. Pemphigus of conjunctiva. Klin. M. f. Augenh., 1925, v. 74, p. 784.

Lundsgaard, K. K. K. Pneumococcus infection of the eye, prophylaxis and treatment. Hospitalstidende, v. 68, p. 505.

Marin Amat, M. Metastatic conjunctivitis of tonsillar origin. Anales de Oft., 1925, v. 1, Feb., pp. 106-108.

Morax, V. Chronic conjunctivitis simulating trachoma. Soc. d'Opht. de Paris, 1925, June, pp. 273-275.

Paderstein, R. What is swimming bath conjunctivitis? Klin. M. f. Augenh., 1925, v. 74, pp. 634-642.

Pratt, J. A. Trachoma. (Dis.) A. J. O., 1925, v. 8, pp. 658-659.

Sedan, J. Contagion of trachoma between infant and nurse. Rev. du Trachome, 1925, v. 2, pp. 72-77.

Segal. Operation of Abadie in trachoma. Paris Thesis, July, 1925. Abst., Gaz. des Hôp., 1925, v. 98, p. 888.

Shinichi Funaishi. Treatment of phlyctenular inflammations of the eye. A. J. O., 1925, v. 8, pp. 618-619.

Singer, J. Parinaud's conjunctivitis. Zeit. f. Augenh., 1925, v. 56, p. 68.

Steiner, L. Follicular conjunctivitis and trachoma. Klin. M. f. Augenh., 1925, v. 74, p. 650.

Tallei, E. Eczematous keratoconjunctivitis. Ann. di Ottal. e Clin. Ocul., 1925, v. 53, p. 764-772.

Terson, A. Technic in cure of trachoma. Rev. du Trachome, 1925, v. 2, pp. 61-72.

CORNEA AND SCLERA.

Best. Cauterization of cornea. Klin. M. f. Augenh., 1925, v. 74, p. 773.

Castresana, A. Examination of corneal degeneration with slitlamp. Anales de Oft., 1925, v. 1, pp. 83-97, Feb.

Derer, J. Gummatous keratitis. Bratislavske Lekar. Listy. June, 1925, pp. 438-443.

Downey, J. W. Corneal disease. A. J. O., 1925, v. 8, p. 649.

Felix, C. H. Congenital family cornea plana. Klin. M. f. Augenh., 1925, v. 74, pp. 710-716.

Filatow, W. P. Keratoplasty. (1 ill.) Klin. M. f. Augenh., 1925, v. 74, pp. 746-748.

Griscom, J. M. Bullous keratitis associated with sinus disease. (Dis.) Atlantic Med. Jour., 1925, v. 28, pp. 740-742.

Koeppe, L. Why transparent layers and membranes of eye are thinner in the axial zone. A. J. O., 1925, v. 8, p. 666.

Kubik. Exudative erythema multiforme. Klin. M. f. Augenh., 1925, v. 74, p. 784.

Levaditi, C. Common origin of epidemic encephalitis and herpes. Paris Méd., 1925, v. 55, p. 573. Abst., J. A. M. A., 1925, v. 85, p. 474.

Lewis, A. J. Discoid keratitis. Med. Jour. of Australia, June 27, 1925, p. 697.

Löhlein, W. Experimental keratitis. (20 ills., bibl.) Arch. f. Augenh., 1925, v. 96, pp. 265-330.

Mans. Epithelial vessels of cornea. Klin. M. f. Augenh., 1925, v. 74, p. 778.

Ohm, J. Treatment of corneal ulcers in coal miners. (10 ills.) Zeit. f. Augenh., 1925, v. 56, pp. 1-21.

Peters. Ocular rosacea. Klin. M. f. Augenh., 1925, v. 74, p. 782.

Robinson, J. Dystrophy of the corneal epithelium. (Dis.) A. J. O., 1925, v. 8, pp. 655-657.

Ring abscess of the cornea. A. J. O., 1925, v. 8, p. 654.

Rossi, D. Traumatic interstitial striate keratitis. Lettura Oft., 1924, Dec., pp. 581-588.

Ruiz Velasco, J. Nitritoid crisis with grave ocular lesions. Rev. de Med. y Cirugia, 1925, v. 30, p. 260. Abst., J. A. M. A., 1925, v. 85, p. 394.

Salus. Rodent ulcer of cornea. Klin. M. f. Augenh., 1925, v. 75, p. 785.

Schlodtmann. Epithelial corneal dystrophy. Klin. M. f. Augenh., 1925, v. 74, p. 777.

Triebenstein. Degeneration of cornea. Klin. M. f. Augenh., 1925, v. 74, p. 777.

Velhagen, K. J. Epithelial scleral implantation cyst. (3 ills., bibl.) Klin. M. f. Augenh., 1925, v. 74, pp. 763-767.

ANTERIOR CHAMBER AND PUPIL.

Carmi, A. Paracentesis of anterior chamber in acute and chronic affections of posterior globe. Lettura Oft., 1925, April, pp. 145-171.

Collin. Persistent pupillary membrane. Soc. d'Opht. de Paris, 1925, June, p. 281.

Hauptvogel. Associated movements of pupil. Klin. M. f. Augenh., 1925, v. 74, p. 785.

Marquez, M. Scheme or plan of pupillary fibers. Anales de Oft., 1925, March, pp. 147-163.

Mazzucconi, M. Measure of pupillary reflex with the Kofman Bujadoux apparatus. (3 ills.) Ann. di Ottal. e Clin. Ocul., 1925, v. 53, pp. 808-814.

Nell. Pupil changes after use of curare, atropin, etc. Pflüger's Arch., 1925, v. 207, pp. 92-103.

Terrien, F. Reaction of Tournoy miosis with abduction. Soc. d'Opht. de Paris, 1925, May, pp. 223-227.

THE UVEAL TRACT.

Back, H. Iridocyclitis and the teeth. Klin M. f. Augenh., 1925, v. 74, p. 783.

Elschnig, A. Etiology of iridocyclitis. Klin. M. f. Augenh., 1925, v. 74, p. 783.

Fuchs, A. Anatomy of inversion and recession of iris. (16 ills., bibl.) A. J. O., 1925, v. 8, pp. 601-608.

Mariotti, C. Adrenalin in hypertensive iritis. Lettura Oft., 1924, Dec., pp. 588-600.

Rollet. Iritis complicating herpes zoster. Lyon Méd, 1925, v. 135, p. 715. J. A. M. A., 1925, v. 85, p. 551.

Sander, E. Acquired detachment of anterior layer of iris. (1 ill.) Klin. M. f. Augenh., 1925, v. 74, pp. 729-730.

Satanowsky, P. Tuberculoma of iris cured with tuberculin therapy. (2 ills.) Semana Med., 1925, v. 32, pp. 29-32.

Thibierge, G. and **Cha''lous, J.** Iritis with hemiatrophy of face. Soc. d'Opht. de Paris, 1925, June, p. 258.

Wendt. Bilateral iridocyclitis and choked disc in serous meningitis. Klin. M. f. Augenh., 1925, v. 74, p. 775.

SYMPATHETIC DISEASE.

Lindemann. Sympathetic ophthalmia and serpent ulcer. Klin. M. f. Augenh., 1925, v. 74, p. 775.

Truc, H. and **Dejean, C.** Sympathetic ophthalmia consecutive to extraction of cataract. (7 ills.) Arch. d'Opht., 1925, v. 42, pp. 385-401.

GLAUCOMA.

Ascher. Comparative value of iridectomy and cyclodialysis. Klin. M. f. Augenh., 1925, v. 74, p. 787.

Bailliart, P. Absorption and loss of fluid of enucleated eye. Soc. d'Opht. de Paris, 1925, June, p. 275.

Blood pressure in the progression of glaucoma. Médecine, Jan., 1925. Abst., Med. Jour. and Record, 1925, v. 122, p. 241.

Basso, D. Sclerectomy in glaucoma. Ann. di Ottal. e Clin. Ocul., 1925, v. 53, pp. 753-763.

Bistis. Primary glaucoma. Arch. d'Opht., 1925, v. 42, pp. 422-426.

Brana, J. Increased ocular tension in sympathotonic patients. (Dis.) Zeit. f. Augenh., 1925, v. 56, p. 68.

Castresana, A. Scleral fistula in Lagrange operation as seen with slitlamp. Anales de Oft., 1925, May, pp. 294-305.

Dieter, W. Osmotic pressure, blood pressure, capillary pressure and ocular pressure. (5 ills.) Arch. f. Augenh., 1925, v. 96, pp. 179-264.

Golowin, S. S. Anterior sclerotomy. (1 ill.) Klin. M. f. Augenh., 1925, v. 74, pp. 737-738.

Goslich, H. A. Preparation for glaucoma operations. Zeit. f. Augenh., 1925, v. 56, p. 40-44.

Jongh, S. E. de and **Wolff, L. K.** Substance which reduces intraocular pressure. Nederland. Tijdsch. v. Geneesk., 1925, v. 1, p. 2665. Abst., J. A. M. A., 1925, v. 85, p. 644.

Lagrange, H. Derangements of organovegetative nervous system and of endocrin in glaucoma. (2 ills., bibl.) B. J. O., 1925, v. 9, pp. 398-408.

Lederer. Influence of radium emanation on intraocular pressure. Klin. M. f. Augenh., 1925, v. 74, p. 785.

Pickard, R. Cavernous optic atrophy and its relation to glaucoma. (13 ills., 1 table.) B. J. O., 1925, v. 9, pp. 385-398.

Samojloff, A. J. Adrenalin in intraocular tension. (6 ills., bibl.) Klin. M. f. Augenh., 1925, v. 74, pp. 652-671.

Schulz. Modification of Elliot operation in Halle clinic. (Dis.) Klin. M. f. Augenh., 1925, v. 74, p. 771.

Seidel, E. Origin, fate and destination of fluids of eye. Deut. med. Woch., 1925, v. 51, p. 851. Abst., J. A. M. A., 1925, v. 85, p. 312.

Siwzew, D. A. Infection after Elliot operation. (5 ills.) Klin. M. f. Augenh., 1925, v. 74, pp. 739-745.

Thiel, R. Physiologic and experimental results in intraocular pressure in healthy and glaucomatous eyes. (13 ills.) Arch. f. Augenh., 1925, v. 96, pp. 331-354.

Voegele, J. Vascular nevus and glaucoma. Klin. M. f. Augenh., 1925, v. 74, p. 775.

Wegner, W. Scotoma with intraocular pressure. Zeit. f. Augenh., 1925, v. 56, pp. 48-52.

CRYSTALLINE LENS.

Barbot. Sclerodermia associated with cataract. Paris Thesis, July, 1925. Abst., Gaz. des Hôp., 1925, v. 98, p. 888.

Barraquer, I. Plea for facoerisis. (4 ills.) Anales de Oft., 1925, v. 1, Jan., pp. 7-14.

Bulson, A. E. Prolapse of iris after cataract extraction. (Dis.) J. A. M. A., 1925, v. 85, pp. 322-325.

Castresana, B. Cataract operation with capsule forceps. (4 ills.) Anales de Oft., 1925, June, pp. 339-360.

Franklin, W. S. and **Cordes, F. C.** Electric cataract. (Dis., bibl.) J. A. M. A., 1925, v. 85, pp. 245-247.

Gifford, S. R. Allergic and toxic properties of lens protein. (Dis.) J. A. M. A., 1925, v. 85, pp. 351-356.

Gourfein-Welt, L. Is the blood serum in cataract eyes different from noncataractous? (Bibl.) Rev. Gén. d'Opht., 1925, v. 39, pp. 147-153.

Graves, B. Slitlamp examination of lens. (Dis.) A. J. O., 1925, v. 8, pp. 650-651.

Lederer. Detachment of Descemet's membrane after cataract operation. Klin. M. f. Augenh., 1925, v. 74, p. 784.

Löwenstein. New theory of senile cataract. Klin. M. f. Augenh., 1925, v. 74, p. 786.

Nichelatti, P. Peripheral iridectomy after Pflüger-Hess in cataract extraction. Lettura Oft., 1925, Feb., pp. 55-71.

Richter, F. Cataract from lightning. Casop. lekar. cesk., 1925, v. 64, p. 821. Abst., J. A. M. A., 1925, v. 85, p. 315.

Smith, A. E. Accommodation after cataract extraction. (Dis.) A. J. O., 1925, v. 8, p. 657.

VITREOUS HUMOR.

Bliedung. Operative treatment of opacities of vitreous. Deut. med. Woch., 1925, v. 51, pp. 1105-1106.

Dejean. Development of vitreous and zonule of Zinn in vertebrates. Paris Thesis, 1924. Rev. Gen. d'Opht., 1925, v. 39, pp. 109-114.

Harbridge, D. F. Intraocular hemorrhage. Southwestern Med., 1925, v. 9, pp. 318-320.

Lebermann, F. Microchemistry of vitreous. (2 ills., bibl.) Arch. f. Augenh., 1925, v. 96, p. 355-384.

THE RETINA.

Abadie, C. Spasm of central artery of retina. Presse Méd., Aug. 1, 1925, pp. 1026-1029.

Antuzzi, C. Consanguinity and retinitis pigmentosa. Lettura Oft., 1924, Sept., pp. 456-459.

Beselin. Retinal disease and optic neuritis. Klin. M. f. Augenh., 1925, v. 74, p. 780.

Chauffard, Grigaut and **Nida.** Insulin treatment of diabetic retinitis. Comp. Rend. de la Soc. de Biolog., 1925, v. 92, p. 1356. Abst., J. A. M. A., 1925, v. 85, p. 392.

Collin. Retinal hemorrhages after contusion of head. Clin. Opht., 1925, v. 29, p. 379-382.

Cucco, A. Transient blindness with spasm of retinal artery. (Bibl.) Ann. di Ottal. e Clin. Ocul., 1925., v. 53, pp. 814-820.

Friedenwald, H. Diabetic retintis. (7 tables, dis.) J. A. M. A., 1925, v. 85, pp. 429-434.

Gescher, J. Physiology of intraocular visible hemorrhage. (2 ills.) Arch. f. Augenh., 1925, v. 96, pp. 419-430.

Greenfield, J. G. and **Holmes, G.** Histology of juvenile amaurotic idiocy. Brain, 1925, v. 48, pp. 183-218.

Hanssen. Origin of retinal detachment. (Dis.) Klin. M. f. Augenh., 1925, v. 74, p. 778.

Heckel, E. B. Fundus changes in conditions of intracranial pressure. (Dis.) A. J. O., 1925, v. 8, pp. 639-649.

Lederer. Retinal edema. Klin. M. f. Augenh., 1925, v. 74, p. 785.

Loomis, E. A. Detachment of retina. (Dis.) A. J. O., 1925, v. 8, pp. 657-658.

Scheerer, R. Circulation phenomenon in retina. Klin. M. f. Augenh., 1925, v. 74, p. 688-700.

Varelmann, H. Hereditary hemeralopia with myopia. (6 charts, bibl.) Arch. f. Augenh., 1925, v. 96, pp. 385-405.

Weintraub, A. Circulation in retinal vessels in leucemic retinitis. (Bibl.) Klin. M. f. Augenh., 1925, v. 74, p. 602.

TOXIC AMBLYOPIA.

Lacat. Treatment of quinin amblyopia. (8 fields.) Clin. Opht., 1925, v. 29, pp. 373-379.

THE OPTIC NERVE

Andre-Thomas and Jumentie. Tumor of the 3rd ventricle and aqueduct of Sylvius. Papilledema and optic atrophy. Soc. de Neurol., June 18, 1925. Gaz. des Hôp., 1925, v. 98, p. 904.

Babonneix. Optic atrophy in hereditary syphilis. Soc. d'Opht. de Paris, 1925, May, pp. 236-246.

Benedetti, S. de. Papillary stasis. (Bibl.) Ann. di Ottal. e Clin. Ocul., 1925, v. 53, pp. 821-831.

Boschi, G. Optic neuritis compared with papillary stasis. Soc. d'Opht. de Paris, 1925, May, pp. 253-255.

Castillo, R. del. Bilateral ectasia of optic papilla. Anales de Oft., 1925, May, pp. 305-312.

Courland. Neuroretinal chronaxia. Paris Thesis, July, 1925. Abst., Gaz. des Hôp., 1925, v. 98, p. 888.

Gibb, J. A. Disease of anterior ethmoid a cause of optic and retrobulbar neuritis. Brit. Med. Jour., 1925, July 4, p. 12.

Goslich, H. A. Optic nerve disease after injury to skull. Zeit. f. Augenh., 1925, v. 56, p. 38.

Heim, H. Irritation therapy in optic nerve atrophy. Med. Klin., 1925, v. 21, p. 1123.

Larssan, S. Choked disc in nephrits. Zent. f. innere Med., 1925, v. 46, p. 434. Abst., J. A. M. A., 1925, v. 85, p. 480.

Mallol de la Riva. Intracranial injections in optic nerve atrophy of luetic origin. Arch. de Oft. Hisp.-Amer., 1925, v. 25, p. 468.

Marin Amat, M. Optic atrophy with lacrimal cyst. Anales de Oft., 1925, March, pp. 175-178.

Meyer, S. J. Choked disc. (Dis.) A. J. O., 1925, v. 8, p. 661.

Neff, E. E. Effect of tryparsamide on optic tract. Wisconsin Med. Jour., 1925, v. 24, p. 120-125.

VISUAL TRACTS AND CENTERS.

Bourguet, J. Psammoma of base of 3rd ventricle with compression of chiasm. Soc. d'Opht. de Paris, 1925, June, pp. 264-271.

Carreras, B. Ocular scotomas in epilepsy. Anales de Oft., 1925, March, pp. 163-175.

Foix Chavany, Hillemand and Schiff-Wertheimer. Obliteration of central artery. Hemianopsia. (1 ill.) Soc. d'Opht. de Paris, May, 1925, pp. 221-223.

Gamble, W. E. and Gamble, R. C. Tuberculosis affecting the optic chiasm. (7 ills., 1 table, dis.) J. A. M. A., 1925, v. 85, pp. 412-416.

Lhermitte and Toupet, J. Psychosensory troubles associated wth hemiplegia. Soc. d'Opht. de Paris, 1925, May, pp. 246-252.

Skotnicki, S. H. Influence of radiotherapy on ocular symptoms in cerebral tumor. (2 ills.) Arch. d'Opht., 1925, v. 42, pp. 402-421.

COLOR VISION.

Ahlenstiel, H. Protanope color comparison in color atlas. Klin. M. f. Augenh., 1925, v. 74, pp. 600-601.

Ammann, E. Color stereoscope. (2 ills.) Klin. M. f. Augenh., 1925, v. 74, pp. 587-600.

Just, G. Heredity of color sense in man. (9 charts.) Arch. f. Augenh., 1925, v. 96, pp. 406-418.

Schiötz, H. J. Flash lantern for testing color sense. (1 ill.) Norsk. Magaz. for Laegevidenskaben., v. 86, pp. 549-552. Klin. M. f. Augenh., 1925, v. 74, pp. 577-581.

Szivare. Ear and color. Klin. M. f. Augenh., 1925, v. 74, p. 799.

Wölfflin, E. Total color blindness with Pulfrisch's stereoeffect. Klin. M. f. Augenh., 1925, v. 74, pp. 581-586.

THE EYEBALL.

Abderhalden. Transplantation of eye. Pflüger's Archiv., 1925, v. 207, pp. 129-133. Klin. M. f. Augenh., 1925, v. 74, p. 794.

Moncrieff, W. F. Congenital coloboma of iris, choroid and optic nerve. A. J. O., 1925, v. 8, p. 662.

Schwartz, V. Congenital anomalies of the eye. (Dis.) A. J. O., 1925, v. 8, pp. 659-660.

Thies. Metastatic ophthalmia after angina. Klin. M. f. Augenh., 1925, v. 74, p. 775.

LACRIMAL APPARATUS.

Bastera Santa-Cruz, J. Technic of Dupuy-Dutemps and Borquet in dacryocystorhinostomy. (14 ills.) Anales de Oft., 1925, April, pp. 211-253.

Casadesus, F. Dacryocystorhinostomy. (8 ills.) Anales de Oft., 1925, v. 1, pp. 98-105.

Dreuschuch and Sacha. Radiography of lacrimal passages. (50 ills., bibl.) Bratislavske Lekar. Listy, 1925, May, pp. 387-405.

Dupuy-Dutemps. Plastic dacryocystorhinostomy. Anales de Oft., 1925, April, pp. 263-267.

Frieberg, T. Some complications in dacryocystorhinostomy. (West.) Dansk. Ophth. Selskabs Forhand., 1924, pp. 29 and 30.

Friede, R. Extirpation of lacrimal gland. Klin. M. f. Augenh., 1925, v. 74, pp. 682-687.

Hessberg, R. Mucous membrane needle in Toti's operation. (1 ill.) Klin. M. f. Augenh., 1925, v. 74, p. 755.

Isakowitz. Folds in tear sac. Klin. M. f. Augenh., 1925, v. 74, pp. 681-682.

Paparcone, E. Surgical treatment of dacryocystitis. Lettura Oft., Feb., 1925, pp. 49-55.

Poyales, F. Dacryocystorhinostomy. (9 ills.) Anales de Oft., 1925, Jan., v. 1, pp. 15-23.

Stock, W. Pathology of the lacrimal organs. (Ill.) Handb. de ges. Augenh. Graefe-Saemisch., 1925, Bd. 9, pp. 1-170.

Triebenstein. Treatment of suppuration of lacrimal sac. Klin. M. f. Augenh., 1925, v. 74, p. 782.

Van der Hoeve, J. and Steenhuis, D. J. Roentgenogram of lacrimal canal after filling with lipiodol. (8 ills.) Klin. M. f. Augenh., 1925, v. 74, pp. 671-681.

DISEASES OF THE LIDS.

Bachmann, R. Tarsorrhaphy and transplantation of fascia in paralytic lagophthalmos. (4 ills.) Klin. M. f. Augenh., 1925, v. 74, pp. 756-762.

Cosmettatos, G. F. Marcus Gunn syndrome (Jaw winking). (2 ills., bibl.) Rev. Gén. d'Opht., 1925, v. 39, pp. 195-200.

Cramer. Streptococcic disease of lids. Klin. M. f. Augenh., 1925, v. 74, p. 773.

Elschnig, A. Gumma of lids. Med. Klin., 1925, v. 21, p. 1079.

Goldenburg, M. Congenital bilateral ptosis. A. J. O., 1925, v. 8, p. 662.

Hanssen, R. Vitiligo. (2 ills.) Zeit. f. Augenh., 1925, v. 56, pp. 35-37.

Kreiker, A. Endogenous tuberculosis of lids. (Bibl.) Klin. M. f. Augenh., 1925, v. 74, pp. 642-649.

Kümmell. Pathologic anatomy of chalazia. (Dis.) Klin. M. f. Augenh., 1925, v. 74, p. 779.

Leal, Junior. Movements of upper lid with mastication (jaw winking). Rev. Gén. d'Opht., 1925, v. 39, p. 158.

Lewis, A. J. Ectropion and symblepharon following trachoma. Med. Jour. of Australia, June 27, 1925, p. 697.

Nizetic, Z. Pustule and carbuncle of lids. (2 ills., bibl.) Arch. d'Opht., 1925, v. 42, pp. 427-438.

DISEASES OF THE ORBIT.

Aszalos, J. Orbital echinococcus. Zeit. f. Augenh., 1925, v. 56, p. 67.

Büttner, G. Pulsating exophthalmos. Beitr. z. klin. Chir., 1925, v. 133, pp. 512-519. Abst., J. A. M. A., 1925, v. 85, p. 395.

Finochietto, R. Operations on the orbit. Rev. de Cirugia, 1925, v. 4, p. 64. Abst., J. A. M. A., 1925, v. 85, p. 311.

Posey, W. C. Orbital abscess in an infant; evacuation thru conjunctiva. A. J. O., 1925, v. 8, pp. 612-613.

Schindler. Orbital swelling. Klin. M. f. Augenh., 1925, v. 74, p. 781.

Torrigiani and Pereyra. Exophthalmos with orbital abscess. Arch. de Oft. Hisp-Amer., 1925, v. 25, p. 452.

Unverricht. Experimental exophthalmos. Ann. di Ottal. e Clin. Ocul., 1925, v. 53, p. 843.

Wright, R. E. and Taylor, C. G. Cellulitis of the orbit. Indian Med. Gaz., 1925, v. 60, pp. 312-316.

INJURIES.

Agnello, F. Complete traumatic aniridia. Lettura Oft., 1925, April, pp. 171-175.

Caldwell, R. Industrial eye injuries. (Dis.) Southern Med. Jour., 1925, v. 18, pp. 619-621.

Cayce, E. B. Supposed eye injury. (Dis.) A. J. O., 1925, v. 8, p. 651.

Ezell, H. Gun shot wound of eyes. (Dis.) A. J. O., 1925, v. 8, pp. 652-653.

Gallois, J. War projectile in orbit tolerated 7 years. Soc. d'Opht. de Paris, 1925, June, p. 280.

Guyer, M. G. Precipitin production thru lens injury. Jour. Inf. Diseases, 1925, v. 37, pp. 93-95.

Huber, W. A. Injuries to the crystalline lens. A. J. O., 1925, v. 8, p. 635.

Lederer. Injury to cornea from chrysarobin. Klin. M. f. Augenh., 1925, v. 74, p. 784.

Löhlein and Studemann. Anilin staining. Klin. M. f. Augenh., 1925, v. 74, p. 774.

Majoli, L. Foreign body in eye nine years. Lettura Oft., 1924, Oct., p. 503.

Mans. Changes in fundus after injury. Klin. M. f. Augenh., 1925, v. 74, p. 776.

Mock. Movement of copper wire in eye. Münch. med. Woch., 1925, v. 72, p. 1076.

Muncaster, S. B. Siderosis bulbi. (1 ill.) A. J. O., 1925, v. 8, pp. 630-632.

Nitsch, M. Extraction of embedded foreign bodies from posterior of eye. (2 ills.) Zeit. f. Augenh., 1925, v. 56, pp. 44-47.

Pischel, K. Copper in eyes and glaucoma. Klin. M. f. Augenh., 1925, v. 74, pp. 651-652.

Tessier, G. Indirect rupture of choroid in contusion injury. Lettura Oft., 1925, March, pp. 118-126.

Vogt, A. Injury of eye by light. Schweiz. med. Woch., 1925, v. 55, p. 425. Abst., J. A. M. A., 1925, v. 85, p. 310.

Woodruff, H. Burned lids and eyeball. (Dis.) A. J. O., 1925, v. 8, p. 662.

TUMORS.

Addario la Ferla, G. Differential diagnosis of glioma and pseudoglioma. Lettura Oft., 1925, March, pp. 103-118.

Belgeri, F. and Lijo Pavia, J. Epithelioma of cornea. (3 ills.) Semana Med., 1925, v. 32, pp. 97-99.

Castillo Ruiz, R. Lymphosarcoma of upper lid. (2 ills.) Anales de Oft., 1925, v. 1, Jan., pp. 23-26.

Cayce, E. B. Melanosarcoma of eye. A. J. O., 1925, v. 8, pp. 651-652.

Elschnig, H. H. Retrobulbar xanthomatous fibroma. Klin. M. f. Augenh., 1925, v. 74, pp. 723-725.

Fischer. Tumor in vitreous. Ann. di Ottal. e Clin. Ocul., 1925, v. 53, p. 843.

Gamble, R. Neoplasms of the eye. (Dis.) A. J. O., 1925, v. 8, pp. 662-664.

Horton, J. J. Neuroepithelioma. (2 ills.) A. J. O., 1925, v. 8, pp. 637-638.

Jaensch, P. A. Blastoma of orbit with carcinomatous disease of parotids. Klin. M. f. Augenh., 1925, v. 74, pp. 716-723.

Kennon, W. G. and Warner, R. J. Intraocular tumor. (Dis.) A. J. O., 1925, v. 8, p. 653.

LoRusso, D. Melanosarcoma of choroid. (4 ills., bibl.) Ann. di Ottal. e Clin. Ocul., 1925, v. 53, pp. 788-793.

Nida and d'Autrevaux, Y. Papilloma of conjunctiva and cornea. (1 ill.) Soc. d'Opht. de Paris, 1925, June, pp. 271-273.

Pfingst, A. O. Glioma of retina. Amer. Med., 1925, July, p. 442.

Satanowsky, P. Primary sarcoma of lid. (13 ills., bibl.) Semana Med., 1925, v. 32, pp. 169-178.

Schwartz, V. Melanosarcoma of the choroid. (Dis.) A. J. O., 1925, v. 8, pp. 660-661.

Wachtler, G. Syphiloma of ciliary body. Klin. M. f. Augenh., 1925, v. 74, pp. 725-729.

PARASITES.

Marquez, M. Intraocular cysticercus. (6 ills.) Anales de Oft., 1925, June, pp. 361-370.

GENERAL PATHOLOGY.

Ford, C. E. Hereditary blindness. J. A. M. A., 1925, v. 85, pp. 378-379.

Odell, A. G. Albinismus. Clifton Med. Bull., 1925, v. 11, pp. 5-10.

GENERAL AND EXTRAOCULAR DISEASES.

Arloing, F., Jung and Lesbates. Oculocardiac reflex in domestic animals. Soc. de Biol., 1925, June 27. Abst., Gaz. des Hôp., 1925, v. 98, p. 918.

Bas, J. Pathogenesis of migrain. Paris Thesis, 1925.

Brown, A. L. Ocular manifestations in serum sickness. (Bibl.) A. J. O., 1925, v. 8, pp. 614-618.

Bouman, L. and Bok, S. T. Experimental encephalitis. Nederland. Tijdsch. v. Genees., 1925, v. 1, p. 2406. Abst., J. A. M. A., 1925, v. 85, p. 644.

Clarke, E. Fundus oculi in general medicine. (Dis.) A. J. O., 1925, v. 8, pp. 664-666.

Finnoff, W. C. Tuberculosis of eye. Texas State Jour. Med., 1925, v. 21, pp. 173-178.

Gourfein, D. Treatment of ocular tuberculosis with bismuth. Rev. Gén. d'Opht., 1925, v. 39, pp. 5-15.

Joshine. Purple vision and vitamines. Ann. di Ottal. e Clin. Ocul., 1925, v. 53, p. 842.

Kuriks, O. Microscopy of ocular leprosy. (1 col. pl.) Klin. M. f. Augenh., 1925, v. 74, pp. 749-755.

Langdon, H. M. Ocular complications of chorea. (Bibl.) A. J. O., 1925, v. 8, pp. 626-629.

Löwenstein. Scrofulous eye disease. Ann. di Ottal. e Clin. Ocul., 1925, v. 53, p. 843.

Majo. Adenoids and ocular affections. Arch. de Oft. Hisp.-Amer., 1925, v. 25, p. 453.

Morelli, E. Leontiasis of cranium and ocular lesions. (4 ills., bibl.) Ann. di Ottal. e Clin. Ocul., 1925, v. 53, pp. 773-787.

Pincus, F. The teeth and the eyes. (3 ills.) Klin. M. f. Augenh., 1925, v. 74, pp. 730-737.

Rosa, G. de. Ocular complications in bubonic plague. Lettura Oft., 1924, Nov., pp. 551-555.

Stübel. Juvenile ocular diseases. Klin. M. f. Augenh., 1925, v. 74, pp. 772.

Vieira, J. Complications in eyes with sinusitis. Brazil Med., 1925, v. 1, p. 259. Abst., J. A. M. A., 1925, v. 85, p. 640.

Van der Hoeve, J. Ocular signs in tuberous sclerosis of cerebral convolutions and multiple neurofibromatosis. Soc. d'Opht. de Paris, 1925, May, pp. 211-221.

VISUAL HYGIENE AND PROPHYLAXIS.

Batten, R. D. The eye from infancy to old age. Practitioner, 1925, v. 115, pp. 63-68.

Belling, J. Compensating the unemployed eye in monocular instruments. Science, 1925, v. 42, p. 54.

Bonner, W. F. Hygiene of the eye. National Safety News, March, 1925, pp. 10-11.

Cramp, A. J. Tinkering with the eyes. Hygeia, 1925, v. 3, pp. 437-439.

Ferree, C. E. and Rand, G. Ocular principles in lighting. Reprint No. 14 from the Illuminating Engin. Soc., p. 22. Abst., Jour. Industrial Hyg., 1925, v. 7, p. 148.

Ives, J. E. Effect of one degree of illumination on working speed in postoffice. Pub. Health Rep., Reprint No. 973, 1924.

OPHTHALMIC SOCIOLOGY.

Award for enucleation of blind eye. J. A. M. A., 1925, v. 84, p. 1689.

Carlotti. Trachoma clinic in Corsica. Rev. du Trachome, 1925, v. 2, April, pp. 77-80.

Fitting glasses by mail. J. A. M. A., 1925, v. 85, pp. 286-289.

Gouzien, P. Control of trachoma in the French Colonies. Rev. du Trachome, 1925, v. 2, Jan., pp. 1-6.

Institute of British ophthalmic opticians. Lancet, Aug. 1, 1925, pp. 224-226.

Jackson, E. Oculist or optometrist? A. J. O., 1925, v. 8, pp. 668-669.

Vision and earning ability. A. J. O., 1925, v. 8, pp. 667-668.

Kostitch, D. Trachoma in Serbia. Rev. du Trachome, 1925, v. 2, April, pp. 80-97.

LaRue, C. L. Ophthalmologist or optometrist. Colorado Med., 1925, v. 22, p. 260.

Loss of sight in operating moving picture machines. (Medicolegal.) J. A. M. A., 1925, v. 85, p. 542.

Oculists and optometrists. J. A. M. A., 1925, v. 85, p. 272.

Patton, J. M. Oculists or optometrists which? (Dis.) J. A. M. A., 1925, v. 85, pp. 564-569.

Schousboe. Prophylaxis of trachome in North Africa. Rev. du Trachome, 1925, v. 2, Jan., pp. 15-27.

Trachoma in the State of Indiana. Jour. Ind. State Med. Assn., 1925, v. 18, pp. 264-265.

EDUCATION, HISTORY AND INSTITUTIONS.

Bach, E. C. Eye clinics of Vienna. Wisconsin Med. Jour., 1925, v. 24, pp. 134-137.

Chance, B. First book printed on diseases of the eye. A. J. O., 1925, v. 8, pp. 638-639.

Morax, V. French School of ophthalmology and neurology. (1 ill.) Soc. d'Opht. de Paris, 1925, May, pp. 204-211.

Twyman, F. Contribution of optical instrument makers to medical science. Lancet, July 25, 1925, p. 209.